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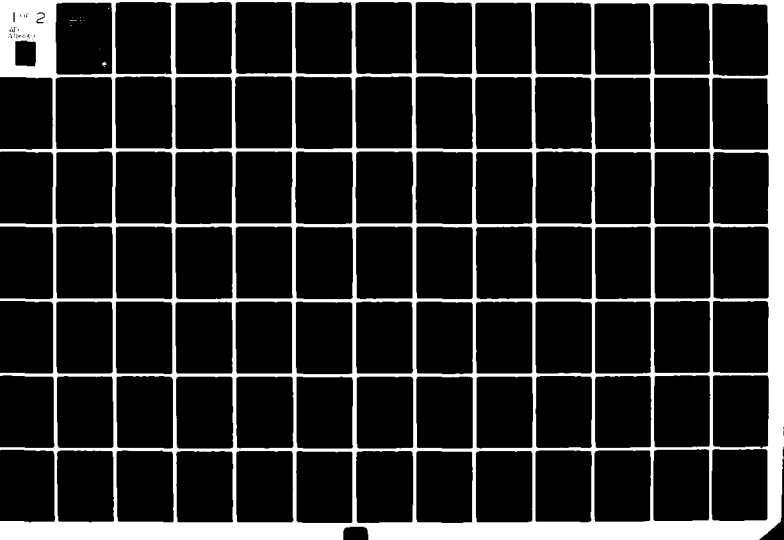

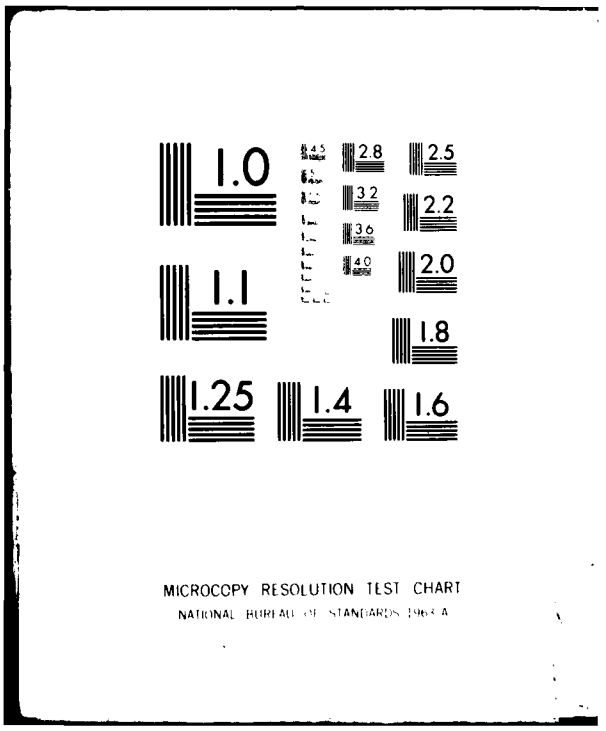
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Report SAM-TR-82-16

USAFSAM REVIEW AND ANALYSIS OF RADIOFREQUENCY RADIATION BIOEFFECTS LITERATURE: SECOND REPORT

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333 Ravenswood Avenue
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May 1982

Final Report for Period 1 September 1980 – 30 June 1981

Prepared for
USAF SCHOOL OF AEROSPACE MEDICINE
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas 78235

NOTICES

This final report was submitted by SRI International, 333 Ravenswood Avenue, Menlo Park, California, under contract F33615-80-C-0608, job order 7757-01-73, with the USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, Brooks Air Force Base, Texas. James H. Merritt (USAFSAM/RZP) was the Laboratory Project Scientist-in-Charge.

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This report has been reviewed by the Office of Public Affairs (PA) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

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Biological effects

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The objectives of this project were to acquire, review, and analyze, on an ongoing basis, information on research pertaining to the biological effects of radiofrequency radiation (RFR), for the USAF School of Aerospace Medicine (USAFSAM). The method used was to: (1) select documents that are judged to be representative of prior and current research on various RFR-bioeffects topics, (2) analyze in detail the contents of each such document, and

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20. ABSTRACT (Continued)

(3) assess the validity and the significance of the results presented. In this report, the major RFR-bioeffects topics are listed, and the format used for analyzing each selected document is described. The text of each analysis completed was prepared and submitted to USAFSAM in optical character recognition (OCR-B) font, to permit direct storage of the information in a computer at USAFSAM. During the contract period, 80 analyses were completed. In the Appendix, these analyses are listed alphabetically by first author under the pertinent major RFR-bioeffects topics, and the texts of analyses not included in the first interim technical report (SAM-TR-81-24) are presented. The Appendix also contains a master list or bibliography of all the analyses completed, arranged alphabetically by first author without regard to topic heading.

CONTENTS

FIGURES

USAFSAM REVIEW AND ANALYSIS OF RADIOFREQUENCY RADIATION BIOEFFECTS LITERATURE: SECOND REPORT

INTRODUCTION

The objectives of this project were to acquire, review, and analyze, on an ongoing basis, information on research pertaining to the biological effects of radiofrequency radiation (RFR), and to provide periodic technical reports of our findings and assessments to the USAF School of Aerospace Medicine (USAFSAM) in specified formats.

The first interim technical report for the period from 1 March through 31 August 1980 was reproduced and issued as SAM-TR-81-24. The present report is for the period from 1 September 1980 through 30 June 1981.

METHODOLOGY

Thousands of scientific papers, reports, books, summaries, and abstracts (referred to collectively as "documents") have been published on the bioeffects of RFR and related fields. Because references to most of these documents are readily available through the various abstracting services, we endeavored to avoid needless duplication of such services. Instead, the approach taken was to: (1) select documents judged to be representative of prior and current research on various RFR-bioeffects topics, (2) analyze in detail the contents of each such document, and (3) assess the validity and the significance of the results presented.

A major aspect of the project was to prepare the analysis of each document selected in a format that permits easy storage of the information in a computer at USAFSAM and retrieval of each entire analysis by means of any of several coded designators, e.g., by major topic, frequency, modulation (pulsed or CW), species. By this means, USAFSAM can use any specific designator code to search for and retrieve all of the analyses pertinent to that designator. During the project, the text of each analysis was prepared and submitted to USAFSAM in an optical character recognition (OCR-B) font, to permit direct storage of the information without retyping. The software for subsequent retrieval is being developed by USAFSAM.

The list of the major topics used (selected by agreement with USAFSAM) is shown in Figure 1. The numerical designator preceding each topic was assigned arbitrarily for coding purposes. Some topics include appropriate, more specific subheadings.

1 Epidemiologic
2 Mutagenic and cytogenetic
3 Teratogenic and developmental abnormalities
4 Ocular
5 Nervous system
6 Behavioral
7 Endocrinological
8 Immunological
9 Biochemical/physiological
10 Cellular
11 Mechanisms of interaction
12 Environmental
13 Medical applications
14 Review
15 Ecological
16 Physical methods/dosimetry
17 Other
18 Drug interactions

Figure 1. Type of study.

The basic outline form used for analyzing each document is shown in Figure 2. The authors, title, and publication citation are given in one of the formats commonly used. In addition, if an International Standard Serial Number (ISSN) has been assigned to the document and is available, it is included with the citation. However, it should be noted that the manner in which ISSNs are assigned varies with the publication. To illustrate this point, the paper by O. P. Gandhi, "State of the Knowledge for Electromagnetic Absorbed Dose in Man and Animals," Proc. IEEE, Vol. 68, No. 1, pp. 24-32 (Jan 1980), has been assigned the ISSN 0018-9219/80/0100-0024, in which the first eight digits represent the journal and the next two the year of the issue. The next four digits represent the number or month of the issue and the last four the first page of the paper. Similarly, the paper by A. W. Guy et al., "Circularly Polarized 2450-MHz Waveguide System for Chronic Exposure of Small Animals to Microwaves," Radio Sci., Vol. 14, No. 6S, pp. 63-74 (Nov-Dec 1979), has been assigned the ISSN 0048-6604/79/1112-S010, in which the first ten digits have the same meaning as before. However, the next four represent the months rather than the issue number, and the last group of four symbols indicates that the issue is a supplemental one and that the paper is the tenth one in the issue (rather than the first page of the paper).

For each document reviewed, one or more pertinent major topics, preceded by their numerical designators (from Figure 1) are listed under "Study type." All important relevant topics are included to ensure retrieval under any one of them. If a subheading is appropriate, it is also shown, e.g., (5) Nervous system (calcium efflux). In addition, whether an experimental investigation was performed in vivo or in vitro and the species studied are also shown under this heading.

Authors:
Title:
ISSN and citation:
Study type: (code and topic; in vivo/in vitro; species)
Effect type:
Frequency/wavelength:
Modulation:
Power densities:
SAR:
Exposure conditions:
Author abstract or reviewer summary:
Other information:
Initial or final critique:
References:

Figure 2. Outline form for analyses.

The specific effects, phenomena, biological endpoints, or other characteristics sought or studied are listed under "Effect type," e.g. "thresholds for auditory perception of RFR." The next four headings are self-explanatory. Information such as duration of RFR exposure, type of exposure facility, RFR characteristics, and other pertinent data is given under "Exposure conditions."

As part of each analysis, a verbatim reproduction of the abstract provided by the authors was included if the document contained one, and the heading "Author abstract" was used to indicate this fact. Under "Other information," any important information in the text of the document that was not included in the abstract was summarized, and previous work by the authors or research by others on the same topics was cited, if pertinent, but no comments on such information were made. If the document did not contain an abstract, its important contents were summarized without comment, and this fact was indicated by using the heading "Reviewer summary." In addition, any pertinent information not appropriate for the summary was presented under "Other information."

If the document contained sufficient information to permit it, a detailed critique of its contents was performed and the result was presented under the heading "Final critique." To the extent possible or appropriate, each critique includes analysis and evaluation of: the data presented, the biological and engineering methodology used, the validity of the results, how the findings compare with those of other investigations, and the significance of the findings with respect to the health of humans (and/or other species) exposed to RFR. If the document contained information of sufficient importance to merit review, but lacked basic information needed for an adequate critique, that fact was stated and the heading "Initial critique" was used, with a view toward seeking or awaiting the additional information necessary for a final critique.

Any other documents alluded to as part of the analysis were listed under "References."

The analyses in the Appendix to this report illustrate this methodology.

PROGRESS DURING THE SECOND PERIOD

By the end of the second period, 80 complete analyses had been performed, which were delivered in final format (OCR-B) for comment and/or storage in the USAFSAM computer. Of these, 49 were included in SAM-TR-81-24 and the remaining 31 are contained in the present report.

The analyses completed to date in final format are listed under their respective topics in the Appendix. Each topic list is followed by the texts of the analyses completed during the second period. Analyses of documents pertaining to more than one topic are listed under all appropriate topics but are not duplicated. Instead, reference is made to the topic under which the analysis is included.

Initially, it was intended to prepare collective summaries of the current state of knowledge of each major topic, based on the analyses completed on that topic. This task was done for "Epidemiologic," and the collective summary was included in SAM-TR-81-24. However, it was subsequently realized that periodic topic summaries based solely on the analyses completed to date on each topic could constitute unbalanced accounts of the overall status of such topics at that time because important documents not yet fully analyzed may not have been included. Moreover, under other projects, SRI has prepared what are believed to be balanced accounts of the then-current status of each topic as parts of Environmental Impact Statements for several proposed RFR-emitting systems. For this reason, the preparation of such collective summaries in this project was discontinued.

PROPOSED PLANS FOR THE FUTURE

It is proposed to continue performing detailed analyses of important publications, reports, books, abstracts, and symposia presentations on biological effects of RFR to augment the data produced thus far under this project.

ACKNOWLEDGMENTS

The contributions of Dr. John S. Krebs (deceased) to this project are gratefully acknowledged.

Dr. Peter Polson, consultant to SRI International, contributed significantly to this report by preparing a number of analyses and reviewing others.

APPENDIX

LISTS AND ANALYSES OF SELECTED DOCUMENTS ON BIOLOGICAL EFFECTS OF RADIOFREQUENCY RADIATION

CONTENTS*

*The number in parentheses preceding each topic is the classification under which the topic is filed.

CONTENTS (Concluded)

DESCRIPTION

The analyses completed during this project are listed in this appendix by major RFR-bioeffects topics in the order shown in Figure A-1, reproduced for convenience from Figure 1 of the main body of this report.

The heading of each topic is preceded by its numerical designator (from Figure A-1). Under each heading, the pertinent analyses completed are listed in alphabetical order by first author. The name of the first author of each document listed is preceded by the superscript 1 or 2, the former to indicate that the analysis of that document was included in the first interim technical report (SAM-TR-81-24) and the latter to signify that the analysis is included in the present report. Analyses of documents pertaining to more than one topic heading are listed under all appropriate headings. However, the text of each such analysis is included under only one topic heading and is listed under the other pertinent topics with a reference to the topic heading under which the text may be found.

To illustrate the procedure above, the paper by Appleton et al., "Microwave Lens Effects in Humans" (1975), is listed under both (1) Epidemiologic and (4) Ocular. The text of the analysis can be found under the latter heading in SAM-TR-81-24, as indicated by the superscript 1 preceding the name "Appleton," and the analysis is also listed under (1) Epidemiologic with the notation: (See "Ocular" for analysis).

Following the list of analyses under each topic are the texts of the pertinent analyses not included elsewhere, i.e., under other topic headings and/or in SAM-TR-81-24.

In addition to the lists of analyses by topic headings, this appendix contains a master list or bibliography of all analyses completed to date, arranged alphabetically by first author without regard to topic headings. Again, the superscript 1 preceding the name of the first author indicates that the analysis may be found in SAM-TR-81-24 and the superscript 2 that the analysis is included in the present report.

1 Epidemiologic
2 Mutagenic and cytogenetic
3 Teratogenic and developmental abnormalities
4 Ocular
5 Nervous system
6 Behavioral
7 Endocrinological
8 Immunological
9 Biochemical/physiological
10 Cellular
11 Mechanisms of interaction
12 Environmental
13 Medical applications
14 Review
15 Ecological
16 Physical methods/dosimetry
17 Other
18 Drug interactions

Figure A-1. Type of study.

(1) EPIDEMIOLOGIC

List of Analyses

[1] Appleton, B., S. Hirsh, R. O. Kinion, M. Soles, G. C. McCrossan, and R. M. Neidlinger, MICROWAVE LENS EFFECTS IN HUMANS Arch. Ophthalmol; Vol. 93, pp. 257-258 (1975) (See "Ocular" for analysis.)

[1] Burdeshaw, J. A. and S. Schaffer FACTORS ASSOCIATED WITH THE INCIDENCE OF CONGENITAL ANOMALIES: A LOCALIZED INVESTIGATION Final Report, Report No. XXIII, 24 May 1973-31 March 1976, Contract No. 68-02-0791, EPA 600/1-77-016 (March 1977)

[1] Cleary, S. F. and B. S. Pasternack LENTICUAR CHANGES IN MICROWAVE WORKERS--A STATISTICAL STUDY Arch. Environ. Health, Vol. 12, pp. 23-29 (1966) (See "Ocular" for analysis.)

[1] Cleary, S. F., B. S. Pasternack, and G. W. Beebe CATARACT INCIDENCE IN RADAR WORKERS Arch. Environ. Health, Vol. 11, pp. 179-181 (1965) (See "Ocular" for analysis.)

[1] Cohen, B. H., A. M. Lilienfeld, S. Kramer, and L. C. Hyman PARENTAL FACTORS IN DOWN'S SYNDROME-RESULTS OF THE SECOND BALTIMORE CASE-CONTROL STUDY In E. G. Hook and I. H. Porter (eds.), POPULATION GENETICS-STUDIES IN HUMANS, Academic Press, New York, pp. 301-352 (1977)

[1] Kalyada, T. V., P. P. Fukalova, and N. N. Goncharova BIOLOGIC EFFECTS OF RADIATION IN THE 30-300 MHZ RANGE In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 52-57 (1974)

[1] Klimkova-Deutschova, E. NEUROLOGIC FINDINGS IN PERSONS EXPOSED TO MICROWAVES In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 268-272 (1974)

(1) EPIDEMIOLOGIC

List of Analyses (continued)

[2]Lancranjan, I., M. Maicanescu, E. Rafaila, I. Klepsch, and H. I. Popescu
GONADIC FUNCTION IN WORKMEN WITH LONG-TERM EXPOSURE TO MICROWAVES
Health Phys., Vol. 29, pp. 381-383 (1975) (See "Endocrinological" for analysis.)

[1]Lilienfeld, A. M., J. Tonascia, S. Tonascia, C. H. Libauer, G. M. Cauthen, J. A. Markowitz, and S. Weida
FOREIGN SERVICE HEALTH STATUS STUDY: EVALUATION OF HEALTH STATUS OF FOREIGN SERVICE AND OTHER EMPLOYEES FROM SELECTED EASTERN EUROPEAN POSTS
Final Report, July 31, 1978, Contract No. 6025-619073, Dept. of Epidemiology, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, MD (1978)

[1]Pazderova, J.
WORKERS' STATE OF HEALTH UNDER LONG-TERM EXPOSURE TO ELECTROMAGNETIC RADIATION IN THE VHF BAND (30-300 MHz)
Pracovni Lekarstvi (in Czech), Vol. 23, No. 8, pp. 265-271 (1971). English translation: JPRS No. UDC 616-001.228.1-057-07 (1971)

[1]Pazderova, J., J. Pickova, and V. Bryndova
BLOOD PROTEINS IN PERSONNEL OF TELEVISION AND RADIO TRANSMITTING STATIONS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 281-288 (1974)

[1]Peacock, P. B., J. W. Simpson, C. A. Alford, Jr., and F. Saunders
CONGENITAL ANOMALIES IN ALABAMA
J. Med. Assoc. Ala., Vol. 41, No. 1, pp. 42-50 (1971)

[1]Robinette, C. D. and C. Silverman
CAUSES OF DEATH FOLLOWING OCCUPATIONAL EXPOSURE TO MICROWAVE RADIATION (RADAR) 1950-1974
In D. G. Hazzard (Ed.), SYMPOSIUM ON BIOLOGICAL EFFECTS AND MEASUREMENT OF RADIOFREQUENCY/MICROWAVES, Dept. of Health, Education, and Welfare, Washington, D.C., HEW Publication No. (FDA) 77-8026 (1977)

(1) EPIDEMIOLOGIC

List of Analyses (continued)

[1]Sadchikova, M. N.
CLINICAL MANIFESTATIONS OF REACTIONS TO MICROWAVE IRRADIATION IN VARIOUS OCCUPATIONAL GROUPS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 261-267 (1974)

[1]Siekierzynski, M.
A STUDY OF THE HEALTH STATUS OF MICROWAVE WORKERS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 273-280 (1974)

[1]Sigler, A. T., A. M. Lilienfeld, B. H. Cohen, and J. E. Westlake
RADIATION EXPOSURE IN PARENTS OF CHILDREN WITH MONGOLISM (DOWN'S SYNDROME)
Bull. Johns Hopkins Hosp., Vol. 117, pp. 374-395 (1965)

(2) MUTAGENIC AND CYTOGENETIC

List of Analyses

[1] Blackman, C. F., M. C. Surles, and S. G. Benane
THE EFFECT OF MICROWAVE EXPOSURE OF BACTERIA: MUTATION INDUCTION
In C. C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Dept. of Health, Education, and Welfare, Washington, D. C., HEW Publication (FDA) 77-8010, pp. 406-413 (1976)

[1] Cohen, B. H., A. M. Lilienfeld, S. Kramer, and L. C. Hyman
PARENTAL FACTORS IN DOWN'S SYNDROME-RESULTS OF THE SECOND BALTIMORE CASE-CONTROL STUDY
In E. G. Hook and I. H. Porter (eds.), POPULATION GENETICS-STUDIES IN HUMANS, Academic Press, New York, pp. 301-352 (1977) (See "Epidemiologic" for analysis.)

[1] Dutta, S. K., W. H. Nelson, C. F. Blackman, and D. J. Brusick
LACK OF MICROBIAL GENETIC RESPONSE TO 2.45-GHZ CW AND 8.5- TO 9.6-GHZ PULSED MICROWAVES
0022-2739/79/0009-0275, J. Microwave Power, Vol. 14, No. 3, pp. 275-280 (1979)

[1] Hamnerius, Y., H. Olofsson, A. Rasmuson, and B. Rasmuson
A NEGATIVE TEST FOR MUTAGENIC ACTION OF MICROWAVE RADIATION IN DROSOPHILA MELANOGASTER
0165-1218/79/0011-0217, Mutation Res., Vol. 68, No. 2, pp. 217-223 (1979)

[2] Huang, A. T., M. E. Engle, J. A. Elder, J. B. Kinn and T. R. Ward
THE EFFECT OF MICROWAVE RADIATION (2450 MHZ) ON THE MORPHOLOGY AND CHROMOSOMES OF LYMPHOCYTES
Radio Sci., Vol. 12, No. 6S, pp. 173-177 (1977) (See "Immunological" for analysis.)

[1] Pay, T. L., E. C. Beyer, and C. F. Reichelderfer
MICROWAVE EFFECTS ON REPRODUCTIVE CAPACITY AND GENETIC TRANSMISSION IN DROSOPHILA MELANOGASTER
J. Microwave Power, Vol. 7, No. 2, pp. 75-82 (1972)

(2) MUTAGENIC AND CYTOGENETIC

List of Analyses (continued)

[1]Sigler, A. T., A. M. Lilienfeld, B. H. Cohen, and J. E. Westlake
RADIATION EXPOSURE IN PARENTS OF CHILDREN WITH MONGOLISM (DOWN'S SYNDROME)
Bull. Johns Hopkins Hosp., Vol. 117, pp. 374-395 (1965) (See "Epidemiologic" for analysis.)

[2]Stodolnik-Baranska, W.
THE EFFECTS OF MICROWAVES ON HUMAN LYMPHOCYTE CULTURES,
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 189-195 (1974) (See "Immunological" for analysis.)

[1]Varma, M. M., and E. A. Traboulay, Jr.
EVALUATION OF DOMINANT LETHAL TEST AND DNA STUDIES IN MEASURING MUTAGENICITY CAUSED BY NON-IONIZING RADIATION
In C. C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Dept. of Health, Education, and Welfare, Washington, D. C., HEW Publication (FDA) 77-8010, pp. 386-396 (1976)

[1]Varma, M. M., E. L. Dage, and S. R. Joshi
MUTAGENICITY INDUCED BY NON-IONIZING RADIATION IN SWISS MALE MICE
In C. C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Dept. of Health, Education, and Welfare, Washington, D. C., HEW Publication (FDA) 77-8010, pp. 397-405 (1976)

(3) TERATOGENIC AND DEVELOPMENTAL ABNORMALITIES

List of Analyses

[2] Berman, E., J. B. Kinn, and H. B. Carter
OBSERVATIONS OF MOUSE FETUSES AFTER IRRADIATION WITH 2.45 GHZ MICROWAVES
Health Phys., Vol. 35, pp. 791-801 (1978)

[1] Burdeshaw, J. A. and S. Schaffer
FACTORS ASSOCIATED WITH THE INCIDENCE OF CONGENITAL ANOMALIES: A LOCALIZED INVESTIGATION
Final Report, Report No. XXIII, 24 May 1973-31 March 1976, Contract No. 68-02-0791, EPA 600/1-77-016 (March 1977) (See "Epidemiological" for analysis.)

[2] Carpenter, R. L. and E. M. Livstone
EVIDENCE FOR NONTHERMAL EFFECTS OF MICROWAVE RADIATION: ABNORMAL DEVELOPMENT OF IRRADIATED INSECT PUPAE
IEEE Trans. Microwave Theory Tech., Vol. 19, No. 2, pp. 173-178 (1971)

[2] Chernovetz, M. E., D. R. Justesen, and A. F. Oke
A TERATOLOGICAL STUDY OF THE RAT: MICROWAVE AND INFRARED RADIATIONS COMPARED
Radio Sci., Vol. 12, No. 6S, pp. 191-197 (1977)

[2] Chernovetz, M. E., D. R. Justesen, N. W. King, and J. E. Wagner
TERATOLOGY, SURVIVAL, AND REVERSAL LEARNING AFTER FETAL IRRADIATION OF MICE BY 2450-MHZ MICROWAVE ENERGY
J. Microwave Power, Vol. 10, No. 4, pp. 391-409 (1975)

[2] Dardalhon, M., D. Averbeck, and A. J. Berteaud
DETERMINATION OF A THERMAL EQUIVALENT OF MILLIMETER MICROWAVES IN LIVING CELLS
0022-2739/79/0012-0307, J. Microwave Power, Vol. 14, No. 4, pp. 307-312 (1979) (See "Cellular" for analysis.)

[2] Dietzel, F.
EFFECTS OF ELECTROMAGNETIC RADIATION ON IMPLANTATION AND INTRAUTERINE DEVELOPMENT OF THE RAT
Ann. N.Y. Acad. Sci., Vol. 247, pp. 367-376 (1975)

(3) TERATOGENIC AND DEVELOPMENTAL ABNORMALITIES

List of Analyses (continued)

[2]Hamrick, P. E., D. I. McRee, P. Thaxton, and C. R. Parkhurst
HUMORAL IMMUNITY OF JAPANESE QUAIL SUBJECTED TO MICROWAVE RADIATION DURING EMBRYOGENY
Health Phys., Vol. 33, pp. 23-33 (1977) (See "Immunological" for analysis.)

[2]Lindauer, G. A., L. M. Liu, G. W. Skewes, and F. J. Rosenbaum
FURTHER EXPERIMENTS SEEKING EVIDENCE OF NONTHERMAL BIOLOGICAL EFFECTS OF MICROWAVE RADIATION
IEEE Trans. Microwave Theory Tech., Vol. 22, No. 8, pp. 790-793 (1974)

[2]Liu, L. M., F. J. Rosenbaum, and W. F. Pickard
THE RELATION OF TERATOGENESIS IN TENEBRIO MOLITOR TO THE INCIDENCE OF LOW-LEVEL MICROWAVES
IEEE Trans. Microwave Theory Tech., Vol. 23, No. 11, pp. 929-931 (1975)

[2]McRee, D. I. and P. E. Hamrick
EXPOSURE OF JAPANESE QUAIL EMBRYOS TO 2.45-GHZ MICROWAVE RADIATION DURING DEVELOPMENT
Radiat. Res., Vol. 71, No. 2, pp. 355-366 (1977)

[2]Olsen, R. G. and W. C. Hammer
THERMOGRAPHIC ANALYSIS OF WAVEGUIDE-IRRADIATED INSECT PUPAE
in Abstracts of Open Symposium on the Biological Effects of Electromagnetic Waves, Helsinki, Finland, p. 62 (1978)

[1]Peacock, P. B., J. W. Simpson, C. A. Alford, Jr., and F. Saunders
CONGENITAL ANOMALIES IN ALABAMA
J. Med. Assoc. Ala., Vol. 41, No. 1, pp. 42-50 (1971) (See "Epidemiologic" for analysis.)

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DEVELOPMENTAL EFFECTS OF MICROWAVES ON TENEBRIO: INFLUENCES OF CULTURING PROTOCOL AND OF CARRIER FREQUENCY
0048-6604/79/1112-S027, Radio Sci., Vol. 14, No. 6S, pp. 181-185 (1979)

(3) TERATOGENIC AND DEVELOPMENTAL ABNORMALITIES

List of Analyses (continued)

[2]Rugh, R., E. I. Ginns, H. S. Ho, and W. M. Leach
RESPONSES OF THE MOUSE TO MICROWAVE RADIATION DURING ESTROUS CYCLE AND PREGNANCY
Radiat. Res., Vol. 62, pp. 225-241 (1975)

[2]Smialowicz, R. J., J. B. Kinn, and J. A. Elder
PERINATAL EXPOSURE OF RATS TO 2450-MHZ CW MICROWAVE RADIATION: EFFECTS ON LYMPHOCYTES
0048-6604/79/1112-S022, Radio Sci., Vol. 14, No. 6S, pp. 147-153 (1979) (See "Immunological" for analysis.)

[2]Stavinoha, W. B., A. Modak, M. A. Medina, and A. E. Gass
GROWTH AND DEVELOPMENT OF NEONATAL MICE EXPOSED TO HIGH-FREQUENCY ELECTROMAGNETIC FIELDS
USAF School of Aerospace Medicine, Brooks AFB, Texas; Final Report SAM-TR-75-51 on Contract F41609-74-C-0018, submitted by University of Texas Health Science Center, San Antonio, Texas (1975)

Analyses under

(3) TERATOGENIC AND DEVELOPMENTAL ABNORMALITIES

Berman, E., J. B. Kinn, and H. B. Carter
OBSERVATIONS OF MOUSE FETUSES AFTER IRRADIATION WITH 2.45 GHZ MICROWAVES
Health Phys., Vol. 35, pp. 791-801 (1978)

Study type: (3) Teratogenic and developmental abnormalities, (9) Biochemical/physiological; IN VIVO; MOUSE

Effect type: RFR-induced fetal abnormalities and weight alterations

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 3.4 to 28 mW/cm.2

SAR: 2.0 to 22.2 mW/g

Exposure conditions: 5X5, 7X4, or 3X5 arrays of pregnant mice in far field for 100 min/day for gestational days 1 though 17 at 3.4, 13.6, or 14 mW/cm.2 and for gestational days 6 through 15 at 28 mW/cm.2 in temp. and rel. humidity of 20.2 deg C and 50%, respectively

AUTHOR ABSTRACT: Pregnant CD-1 mice were exposed to 2.45 GHz CW radiation for 100 min daily at a range of power densities (3.4-28 mW/cm.2). Near-term fetuses were examined for gross external morphologic alterations. Mean live fetal weight per litter decreased significantly with exposure to the highest power density (sham, 0.97 plus or minus 0.15 g; irradiated, 0.89 plus or minus 0.13 g). There was a significantly increased incidence of cranioschisis in exposed fetuses. An exposure of the dam for 100 min at these power densities did not appear to be significant thermally. Estimates of mean dose rate as determined using twin-well calorimetry ranged from 2.0 to 22.2 mW/g.

OTHER INFORMATION: SARs were measured by twin-well calorimetry for each array position. For 5X5 arrays irradiated at 10 mW/cm.2, the mean SAR varied with position from 4.05 to 7.37 mW/g, with an array mean of about 5.9 mW/g, yielding 2.0 mW/g at 3.4 mW/cm.2 and 8.1 mW/g and 13.6 mW/cm.2. The array mean for the 3X5 arrays was 22.2 mW/g at 28/cm.2. Mean rectal temperatures decreased slightly after exposure or sham-exposure except for the group exposed at 28 mW/cm.2, for which the mean value after exposure was non-significantly higher than the pre-exposure value. All mice were euthanized on day 18 and their uteri were examined for the number of resorbed and dead

conceptuses and live fetuses. The live fetuses were examined for gross morphological alterations and weighed. At 3.4, 13.6, 14, and 28 mW/cm.2, the numbers of litters treated were 103, 109, 62, and 44, respectively; the corresponding numbers of sham-exposed litters were 117, 106, 73, and 40. Ten types of anomalies were tabulated by the numbers of litters affected. (The numbers of fetuses affected in each litter were not presented.) A total of 27 of the 318 RFR-exposed litters, irrespective of power density, had one or more live abnormal fetuses versus 12 of the 336 sham-exposed litters. For most of the individual anomalies, the numbers of litters affected were either too small for statistical treatment or no RFR-related pattern was apparent. As an example of the latter, 4 litters exposed at 3.4 mW/cm.2 exhibited hematoma, with none in the corresponding sham-exposed group; however, 2 litters exposed at 13.6 mW/cm.2 and 3 sham-exposed litters were affected, and no litters were affected at 14 or 28 mW/cm.2 whereas 1 litter each of their corresponding controls was. By contrast, cranioschisis (akin to exencephaly or brain hernia) was exhibited by 7 litters exposed to RFR, i.e., by 1 litter each at 3.4 and 13.6 mW/cm.2, 3 at 14 mW/cm.2, and 2 at 28 mW/cm.2, and by none of the control groups. However, there is no apparent pattern relating these numbers to power density. The authors indicate that the number at each power density was not significantly different from zero, but that their sum over all power densities (7 of 318 RFR-exposed litters versus none of 336 sham-exposed litters) was significant. The mean live fetal weights of the litters exposed at 3.4, 13.6, and 14 mW/cm.2 were not significantly different from those of the corresponding sham-exposed litters; however, the mean weight for the litters treated at 28 mW/cm.2 was significantly lower than for the sham-exposed litters.

FINAL CRITIQUE: The large positional variations of mean SAR (e.g., from 4.05 to 7.37 mW/g in 5X5 arrays exposed at 10 mW/cm.2) may be an indication of mutual RFR interactions among the mice. The authors correctly state that despite such variations, there was no overlap of SAR between arrays exposed at 13.6 and 3.4 mW/cm.2, corresponding to a power density ratio of 4:1. However, no SAR distribution data were given for the 3X5 arrays exposed at 28 mW/cm.2 or the 7X4 arrays exposed at 14 mW/cm.2, for which the power density ratio was only 2:1, thereby raising the question of possible SAR overlap in these experiments. Regarding abnormal fetuses, statistical treatment of the numbers of litters rather than the numbers of fetuses affected is of questionable validity. Also questionable is the summation of all litters exhibiting cranioschisis (irrespective of power density) and ascribing the statistically significant result to RFR exposure. Taken together, nevertheless, the

results indicate that the levels of RFR used (which were not lethal to the dams) are marginally teratogenic to mice, a conclusion that is consistent with the findings of Rugh et al. (1975), but at variance with the findings of Chernovetz et al. (1975), who concluded that near-lethal levels of RFR (to the dams) were not teratogenic. Differences in exposure arrangements and durations, mouse strains used, gestational days of treatment, and handling of the dams may have contributed to such disparate conclusions. It should be noted that exposure of mice to one 100-min session at SARs from 2 to 22 mW/g corresponds to integrated doses ranging from about 3 to 32 cal/g, which not only include the sublethal range used by Rugh et al. (3 to 8 cal/g) but also values that exceed their average lethality dose (12 cal/g). This point supports the hypothesis that the thresholds derived from the data of Rugh et al. are for dose-rate rather than integrated dose. Another noteworthy point is that Berman et al. exposed their mice daily for the first 17 gestational days rather than only once. Perhaps the only other clearly discernible positive RFR-induced result of this investigation is the smaller mean live fetal weight found for the group exposed at 28 mW/cm.2. The rectal temperature results could be construed as weakly supporting the hypothesis that the latter effect was thermal.

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Carpenter, R. L. and E. M. Livstone
EVIDENCE FOR NONTHERMAL EFFECTS OF MICROWAVE RADIATION: ABNORMAL DEVELOPMENT OF IRRADIATED INSECT PUPAE
IEEE Trans. Microwave Theory Tech., Vol. 19, No. 2, pp. 173-178 (1971)

Study type: (3) Teratogenic and developmental abnormalities; IN VIVO; TENEBRIO MOLITOR (DARKLING BEETLE)

Effect type: Abnormalities in emergent adult beetles due to RFR exposure of pupae

Frequency/wavelength: 10 GHz

Modulation: CW

Power Densities: Not stated; estimated as about 68 and 17 mW/cm.2

SAR: Not stated; estimated as about 40 mW/g for 20 mW

Exposure conditions: Individual pupae in waveguide, with long axis of pupa parallel to waveguide axis and head toward source. Waveguide terminated and matched with pupa inserted. Exposures were for 20 or 30 min at 80 mW (about 68 mW/cm.2) or 2 hrs at 20 mW

AUTHOR ABSTRACT: Several investigators have reported experiments in which microwave radiation caused biological damage at tissue temperatures which were not harmful when brought about by means other than microwaves. To study the effects of 10-GHz CW radiation on a poikilothermic invertebrate animal, we irradiated early pupae of the mealworm beetle, Tenebrio molitor. Each pupa was inserted in a waveguide and irradiated therein at waveguide powers of 80 mW for either 20 or 30 min or at 20 mW for 120 min, after which their subsequent development was observed. In control groups similarly treated, except that no microwave power was applied, 90 percent metamorphosed to become normal adult beetles. In the irradiated groups only 24 percent developed normally. In half of the abnormal animals, the front half had undergone metamorphosis to form a normal beetle head and thorax but the hind part remained in the pupal state. Temperature increases within pupae were recorded during irradiation. When these thermal conditions were duplicated by means of radiant heating, subsequent development of pupae was normal in 80 percent of the experiments. We therefore concluded that the abnormalities induced by microwave radiation were not a thermal effect.

OTHER INFORMATION: The authors briefly review experimental results of other investigators indicative of nonthermal RFR effects. In their own work, pupae in the first or second day of pupation were placed in styrofoam blocks and exposed individually to 10.155-GHz RFR, after which each was returned to its glass vial to develop. Another group of pupae was sham-exposed, and a third group was allowed to develop in individual glass vials without any manipulation or treatment during pupation. All pupae were kept at room temperature. Following emergence, each adult beetle was assigned to one of the following categories: (1) death during pupation, (2) abnormal adults (with 3 subcategories), and (3) morphologically normal adults. Of the 137 untreated pupae, 122 (89%) emerged as normal beetles, 8 (6%) were abnormal, and 7 (5%) died during pupation. Of the 15 sham-exposed for 20 min, 14 (93%) were normal and 1 (7%) was abnormal. Of the 29 sham-exposed for 30 min, 26 (90%) were normal, 2 (7%) were abnormal, and 1 (3%) was dead. Thus, about 90% of the control pupae emerged as normal adults. By contrast, of the 80 pupae exposed for 20 min at 80 mW, 19 (24%) adults were normal, 41 (51%) were abnormal, and 20 (25%) were dead. Also, of 35 exposed for 30 min at 80 mW, 12 (34%) were normal, 15 (43%) were abnormal, and 8 (23%) were dead. Lastly, of 25 exposed for 2 hrs at 20 mW, 5 (20%) were normal, 19 (76%) were abnormal, and 1 (4%) was dead. In addition, exposure to RFR prolonged the pupation period by 3 days, and the lives of the abnormal beetles were shorter than those of normal beetles. A thermocouple inserted in the abdomen of a pupa was used to measure temperature during irradiation. At 80 mW, a temperature rise of about 12 deg C was reached in 6 min and remained constant for the rest of the 20-min exposure period; at 20 mW, the rise was about 3 deg C during the first 7 min and remained constant for the balance of the 2-hr exposure period. A controlled-temperature chamber was used to reproduce the same temperature-time conditions in pupae without RFR. Of 20 pupae heated to stimulate 80 mW for 20 min, 15 (75%) emerged as normal beetles and 5 (25%) as abnormal; of another 20 heated to simulate 20 mW for 2 hrs, 17 (85%) were normal and 3 (15%) were abnormal. The authors conclude that abnormal development in RFR-exposed pupae cannot be explained as a thermal effect.

FINAL CRITIQUE: Other investigators, notably Lindauer et al. (1974), Liu et al. (1975), and Green et al. (1979, 1977), have qualitatively confirmed these findings of RFR teratogenesis in Tenebrio molitor. However, the data of these other investigations for both RFR-exposed and control pupae were widely variable, indicative of the presence of uncontrolled factors. For example, Green et al. (1979) found that RFR susceptibility may be higher for pupae exposed and

cultured under conditions of low relative humidity (less than 35%) than for those at higher humidity. Pickard and Olsen (1979) investigated the influence of several possible factors. They found that control pupae derived from larvae obtained from one supplier and maintained on Purina dairy meal developed into fewer abnormal beetles than control pupae derived from three shipments of larvae from another supplier and maintained on Kellogg's Special K, and that the pupae from the former group appeared to be less susceptible to RFR-induced abnormalities than those from the latter three groups. Also, some results from the latter groups were ambiguous, ranging from RFR being doubtfully deleterious statistically to significantly beneficial, with statistically significant differences among the three groups. Nevertheless, Pickard and Olsen (1979) concluded that RFR at the frequencies, dose rates, and exposure durations used is teratogenic to Tenebrio molitor. Olsen and Hammer (1978) determined the spatial distribution of RFR absorption by thermographic imaging of pupae irradiated at 1.3, 5.95, and 10 GHz in waveguide and found large local variations of SAR at power densities comparable to those used in the investigations cited above. Such spatial distributions would not be obtained in pupae heated in an oven (without RFR) or other uniform-temperature environment. Therefore, the hypothesis that such RFR teratogenesis is a nonthermal effect remains unproved.

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Chernovetz, M. E., D. R. Justesen, and A. F. Oke
A TERATOLOGICAL STUDY OF THE RAT: MICROWAVE AND INFRARED RADIATIONS COMPARED
Radio Sci., Vol. 12, No. 6S, pp. 191-197 (1977)

Study type: (3) Teratogenic and developmental abnormalities, (9) Biochemical/physiological;
IN VIVO; RAT

Effect type: RFR- and IR-induced dam mortality, resorptions and fetal anomalies; differences in: percentages of living fetuses, mean fetal mass, and whole-brain norepinephrine and dopamine

Frequency/wavelength: 2.45 GHz

Modulation: 60-Hz-intensity and 12-Hz mode-stirrer modulation

Power Densities: Not stated

SAR: 31 plus or minus 3 mW/g

Exposure conditions: Each pregnant rat was exposed for 20 min to either RFR or IR or sham-exposed on only one day during days 10 through 16 of gestation. RFR was administered in a multimode, mode-stirred cavity, and IR in a forced-air microbial incubator

AUTHOR ABSTRACT: Primigravid rats were exposed for 20 min to microwave radiation (2450 MHz D-dot or SAR = 31 plus or minus 3 mW/g) in a multimode cavity or to infrared radiation in an incubator (ambient temp about 47 deg C). They were exposed during one of seven days of gestation, the 10th through 16th. The mean change of the dams' colonic temperature, about 3.5 deg C, was equated in the microwave and infrared treatments. Control groups of primigravid dams were sham radiated. During the 19th day of gestation, fetuses were taken by Caesarian section and were examined for structural abnormalities and for insult. In addition, fetal brains were analyzed for levels of dopamine (DA) and norepinephrine (NE). Findings: (1) the incidence of lethal dosing was 27% and 12%, respectively, in microwave- and infrared-irradiated dams; (2) the averaged numbers of fetal resorptions in infrared- and sham-irradiated dams are low (both ~ 2%) but the number is elevated nearly sixfold in microwave irradiated dams; (3) the incidence of mortality in nonresorbed fetuses was nil; (4) extensive hemorrhagic signs were observed in both microwave- and infrared-irradiated fetuses but there was no discernible evidence of structural malformation; (5) averaged fetal mass

is slightly (10%) but reliably smaller in both irradiated groups as compared with controls; and (6) averaged whole-brain DA levels do not differ reliably as a function of treatment but the averaged level of NE in brains of microwave irradiated fetuses is reliably below those of infrared and sham-irradiated fetuses. While the averaged temperature changes of the two groups of irradiated dams are equal, increments of temperature of microwave irradiated dams were more variable; correlative analysis provided evidence that mortality and resorption are probably a function of peak body temperature irrespective of source of radiation.

OTHER INFORMATION: Two rats each were sham-exposed in the RFR cavity for 20 min on one day during days 10 through 16 of gestation; of these, 2 were found to be non-gravid, reducing their number to 12 pregnant rats. These rats served as controls for both the RFR and infrared-radiation (IR) exposure groups. Four rats each were exposed to IR on days 10, 11, 14, 15, and 16, and 5 each on days 12 and 13; 4 were non-gravid, leaving 26 rats. Five rats each were exposed to RFR on days 10 and 12, and 4 each on the other gestational days, of which 4 were non-gravid, leaving 26 rats. Thus, the total of pregnant rats was 64. During RFR or sham-exposures, the cavity was maintained to within 10% of 22 deg C and 50% relative humidity by forced ambient air. For IR exposures, the incubator temperature was 47 plus or minus 7 deg C and the relative humidity was between 10 and 15%. This incubator mean temperature was selected to produce approximately equivalent increases in mean colonic temperature as obtained from RFR exposure. The colonic temperature of some of the controls decreased and those of others increased, yielding a weighted mean increase of 0.06 (reported as 0.04) deg C. The increases for the IR and RFR groups were 3.40 (reported as 3.45) and 3.15 (reported as 3.42) deg C, respectively. Three dams died after IR exposure, 7 after RFR exposure, and none in the control group. On day 19 of gestation, the 54 surviving dams were euthanized and the numbers of implantations and resorptions were counted. Also, each fetus was examined for morphological abnormalities and its viability and mass were determined. The percentages of living fetuses per dam were about 98% each for the control and IR groups and 87% for the RFR group, the latter representing a statistically significant decrease. The mean fetal mass for the control groups was 1.63 g, and the values for the IR and RFR groups were 1.53 and 1.54 g, respectively, both significantly lower than for the control group. No structural abnormalities were evident in any of the 468 formed fetuses, which were all alive when taken, but severe edema and hemorrhagic signs were endemic in the IR and RFR groups. The brains of 60 fetuses were assayed for norepinephrine (NE) and

dopamine (DA) in 4 groups each of 5 pooled brains from control, IR-exposed, and RFR-exposed animals. The averaged level of NE for the RFR group was significantly lower than for the controls, but only marginally lower than for the IR group. The averaged levels of DA ranked similarly but the differences were not statistically significant. In their discussion, the authors conclude the following: "Considered in sum, our findings could be taken as evidence that a brief but highly thermalizing application of 2450-MHz microwaves or of infrared energy have biological effects both comparable and different when averaged colonic temperature changes are equal." They then speculate on various aspects of their findings.

FINAL CRITIQUE: Matching of thermal burdens imposed from exposure to IR and RFR sources by equalizing increases in colonic temperature is questionable not only because of differences in penetration depth (discounted by the authors because of blood-circulation heat equalization) but also because thermoregulation renders colonic temperature an insensitive measure of heat stress. Moreover, the relative humidity in the IR incubators (10 to 15%) was considerably lower than in the RFR chamber (45 to 55%). Irrespective of this point, thermal matching of mean colonic temperature rises is virtually meaningless when the variations from rat to rat are relatively large, as was the case here. In fact, though the pattern was not entirely consistent, the data show that the deleterious effects observed were mostly for the rats having large colonic temperature increases, a point alluded to by the authors. (It should also be noted that the thermal matching was not as close as implied by the cited mean colonic temperature increases of 3.45 and 3.42 deg C for the IR and RFR groups, respectively; recalculation of the weighted mean increases from the data presented yielded 3.40 and 3.15 deg C, respectively.) Another major problem with this investigation was the small number of rats involved (a point recognized by the authors), necessitating averaging the data in each group over the 10-16 day gestational period, a questionable procedure both biologically and statistically. Perhaps a minor point was the use of the sham-exposed rats as controls for the IR group instead of a separate set of sham-IR controls, in view of the aforementioned relative humidity difference. Because of such problems, it is difficult to assess the validity of either the positive or negative results of this investigation. It is noted that in a previous investigation (Chernovetz et al., 1975), the authors concluded that a brief but nearly lethal exposure of pregnant mice to 2.45 GHz was not teratogenic.

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J. Microwave Power, Vol. 10, No. 4, pp. 391-409 (1975)

Chernovetz, M. E., D. R. Justesen, N. W. King, and J. E. Wagner
TERATOLOGY, SURVIVAL, AND REVERSAL LEARNING AFTER FETAL IRRADIATION OF MICE BY 2450-MHZ MICROWAVE ENERGY
J. Microwave Power, Vol. 10, No. 4, pp. 391-409 (1975)

Study type: (3) Teratogenic and developmental abnormalities, (6) Behavioral; IN VIVO; MOUSE

Effect type: Increases in fetal mortality and morbidity due to RFR exposure or cortisone injection; alterations in maze performance of pups from dams exposed to RFR or injected with cortisone

Frequency/wavelength: 2.45 GHz

Modulation: 60-Hz-intensity and 12-Hz mode-stirrer modulation

Power Densities: Not stated

SAR: 38 mW/g

Exposure conditions: Groups of 5 pregnant mice were concurrently exposed to RFR for 10 min on only one day during days 11 through 14 of gestation in a multimode, mode-stirred cavity at an ambient temperature of 22 plus or minus 3 deg C and relative humidity of 50 plus or minus 10%

AUTHOR ABSTRACT: In the first of two factorially designed studies, 80 primigravid mice of the C3H-HeJ strain were subjected to 2450-MHz sinusoidally modulated microwave radiation or to sham radiation (with or without an accompanying injection of 5 mg of cortisone as a teratological marker) on the 11th, 12th, 13th, or 14th day of gestation. The radiation treatment consisted of a single intense dosing of microwave energy (38 mW/g for 600 sec. =22.8 J/g) in a multi-mode cavity. On the 19th day of gestation fetuses were taken via Caesarean section and were observed for gross structural abnormalities. While radiation of dams failed reliably to increase the incidence of fetal mortality or morbidity above that of controls, the dams treated with cortisone gave birth to reliably greater numbers of stillborn and deformed fetuses. In the second experiment during their 14th day of gestation 60 primigravid mice received the radiation or sham-radiation treatment, half with, half without, the accompanying injection of cortisone. A virtually complete failure to survive to weaning characterized the pups born of the sham-radiated cortisone-treated group of dams, but the incidence of cortisone-induced mortality was reliably reduced in pups whose dams were also radiated by microwave energy. Pups sampled from

all but the depleted group were observed later as young adults for competency in mastering a series of reversal habits in a water maze. No differences in maze performances were observed in the mice as a function of their placement in the control or the radiation condition, but offspring of cortisone-treated, radiated dams made reliably more errors. Careful measurement of elevations of colonic temperatures of radiated dams shortly after treatment with cortisone revealed an averaged temperature increase that is close to that observed in a comparably radiated volume of water of equivalent mass. If the finding has generality beyond the gravid mouse--if, that is, cortisone effectively and reversibly renders the mammal ectothermic--an important advance in biological dosimetry of non-ionizing radiation may be at hand.

OTHER INFORMATION: In the first study, a group of 5 pregnant mice was exposed concurrently to RFR for 10 min on day 11 of gestation; another group was exposed on day 12, and two other groups were exposed on days 13 or 14, totaling 20 dams. Another 4 groups were similarly sham exposed. In each case, the 5 mice were free to move about within the exposure chamber. Eight other groups were injected with cortisone (a teratogen), half of which were similarly exposed to RFR and the other half were sham exposed. The second exposure regimen was similar to the first except that the treatments were administered only on gestational day 14 and involved only 3 groups of 5 per treatment, totaling 60 dams. Colonic temperature before and after RFR exposure were measured for 19 dams. All mice of the first study were euthanized on gestational day 19, the numbers of implantations and resorptions were counted, and the fetuses were examined for structural abnormalities. The data were treated statistically, with the 10% level as the boundary for significance. There were no statistically significant percentage differences in fetal mortality or structural abnormalities between RFR and sham-exposed groups not administered cortisone, and no dependence on gestational day of treatment; however, the percentage of normal fetuses was 61% for those injected with cortisone and sham exposed, and 50% for the cortisone + RFR groups. These percentages were significantly lower than those for the non-cortisone groups (both 81%), but not significantly different from each other. The mean colonic temperature of 9 dams not injected with cortisone prior to RFR exposure was 38.58 deg C and the post-exposure value was 40.60 deg C. The corresponding values for the 10 injected dams were 34.59 and 39.93 deg C. In the second study, all dams completed term, and the numbers of pups that survived to weaning at post-partum age 21 days were noted. The results for the non-injected groups were 81 pups from those sham-exposed and 93 from those RFR-exposed, a non-significant

difference. From the cortisone-injected groups, the results were 25 pups from those RFR-exposed and only 2 from those sham-exposed (the latter number too small for the behavioral study). These values were significantly lower than those for the non-injected groups, and their difference was also significant. At 38 days of age, 9 pups from the non-injected sham-exposed groups, 15 from the non-injected RFR-exposed groups, and 11 from those that received cortisone and RFR were selected and their ability to learn to swim a cold-water (16 deg C) Lashley-III maze in one direction and to subsequently learn to swim it in the reverse direction (reversal learning) were studied. The motor abilities of all three groups were essentially the same. Also, statistical treatment of the errors made in the original learning habit showed no significant differences between the two non-cortisone groups, but higher error scores for the combined-treatment group. All three groups showed significant changes in their respective error scores for reversal learning. The authors conclude that "Given the physical parameters, experimental conditions, and strain of animal used in our studies, we conclude that the teratogenic potential of a brief but nearly lethal exposure to microwave energy is nil."

FINAL CRITIQUE: RFR exposure of 5 freely-moving mice concurrently could yield considerable variations in dose rate from mouse to mouse because of mutual interactions as well as spatial variations within the microwave cavity, but their movements would tend to reduce such variations. Whatever the unknown individual values of dose rate and total dose, the mean colonic temperature increases (about 2 deg C for the non-injected RFR-exposed dams, and more than 5 deg C for the combined-treatment group) indicates the significant additional thermal burden imposed on these mice. The finding that exposure to RFR alone produced no significant differences in the percentage of normal fetuses whereas injection with cortisone (with and without RFR exposure) yielded significantly fewer normal fetuses would appear to be strong evidence that near lethal levels of RFR are not teratogenic to mice. The numbers of pups that survived to weaning in each group and their learning abilities tend to support this conclusion. However, this conclusion is at variance with that of Rugh et al. (1975), who found that exposure of mouse dams to sublethal doses of RFR was teratogenic. Moreover, exposure for 10 min at 38 mW/g, as done by Chernovetz et al., corresponds to a dose of 5.44 cal/g, which is well within the teratogenic range reported by Rugh et al. It is also noted that the dosage for lethality of 10% of dams, reported by Chernovetz et al. from a pilot study, was about 5.7 cal/g or about half the mean value found by Rugh et al. Among the possible reasons for these apparently

contradictory findings are the respective differences in exposure systems (cavity versus waveguide), the use of multiple versus individual animal exposures, gross uncertanties in actual doses, the mouse-strain difference (C3H/HeJ versus CF1), dam handling, and differences in gestational day of treatment (day 11 through 14 versus day 8). Also, Chernovetz et al. found fetal anomalies in about 20 % of their control mice whereas Rugh et al. apparently used no controls, presumably under the assumption that the natural incidence of exencephaly is rare. Both groups of investigators indicate that extrapolation of their findings to higher mammalian species is an open question subject to experimental validation, with which we concur.

REFERENCES: Rugh, R., E. I. Ginns, H. S. Ho, and W. M. Leach, RESPONSES OF THE MOUSE TO MICROWAVE RADIATION DURING ESTROUS CYCLE AND PREGNANCY, Radiat. Res., Vol. 62, pp. 225-241 (1975).

Dietzel, F.
EFFECTS OF ELECTROMAGNETIC RADIATION ON IMPLANTATION AND INTRAUTERINE DEVELOPMENT OF THE RAT
Ann. N.Y. Acad. Sci., Vol. 247, pp. 367-376 (1975)

Study type: (3) Teratogenic and developmental abnormalities, (13) Medical applications, (10) Cellular; IN VIVO; RAT

Effect type: RFR-induced fetal abnormalities and decrease in DNA synthesis in tumors

Frequency/wavelength: 27.12 and 461 MHz

Modulation: CW

Power Densities: Not measured

SAR: Not measured

Exposure conditions: Abdominal exposure of gravid mice to 27.12 MHz with a diathermy machine and applicator at 55, 70, or 100 W electric power for durations that produced rectal temperatures not exceeding 42 deg C. Acute heating of superficial tumors with 461 MHz to 42 deg C

REVIEWER SUMMARY: 749 rats pregnant with 7800 embryos between gestational days 1 and 16 were abdominally exposed briefly once to 27.12 MHz with a diathermy machine and applicator in 3 experimental groups involving electric powers of 55, 70, and 100 W. The rectal temperature of each rat was monitored with a mercury thermometer during exposure and the rat was removed from the field when the rectal temperature reached a predetermined value for each group in lieu of any other dosimetry. On day 20, the fetuses were removed, counted, weighed, and examined for external malformations. In addition, embryos in resorption and corpora lutea graviditatis were counted, and the preimplantation losses were calcuated by subtracting the numbers of mature and resorbed fetuses from the number of corpora lutea graviditatis. Typical predominant abnormalities included neurocranial malformations from RFR exposure on days 9 and 10, kinked or short tails and hand defects from exposure on days 13 and 14, and cleft palate for day 15. The frequency of external malformations was highest for those exposed on days 13 and 14, and correlated well with maximum rectal temperatures. The values were 0.27% malformations at 39 deg C for a group exposed with 55 W for 5 min, 12.4% at 40.5 deg C for a group exposed with 70 W for 10 min, and 46.1% at 42 deg C, for exposure with 100 W for 10 min. The calculated preimplantation loss was about 55% for days 1

and 2, diminished rapidly to less than 20% for days 7 and 8, and was close to the control value of about 14% for most of the remaining gestational days of exposure. Postimplantation loss (after organogenesis) increased slowly from the 10% control value for exposure during days 1 through 6 and rose rapidly to about 22% for days 15 and 16. The higher values were ascribed to RFR-generated-heat accumulation in the amniotic sac. The effect of tumor treatment with 461 MHz on DNA synthesis, as determined from incorporation of P-32, was compared with treatment with X-rays. Tumor heating to 42 deg C with the RFR decreased the P-32 incorporation rate by about 13% at 2 hrs post-treatment and by about 27% at 12 hrs. The decrease produced by X-ray treatment was negligible at 2 hrs and only about 7% at 12 hrs.

FINAL CRITIQUE: This investigator found that the types and frequencies of fetal anomalies depend on the gestational day of RFR exposure, with maximum frequency on days 13 and 14, in qualitative agreement with the findings of others. The correlation of percentage of anomalies with rectal temperature indicates that the results were due to heating by RFR. However, the lack of RFR dosimetry in this investigation renders it difficult to make quantitative comparisons. Noteworthy are the high calculated percentages of preimplantation losses due to RFR exposure during the early days of gestation. Similar calculations were apparently not made by other investigators. Also interesting was that the postimplantation loss was substantially constant at control values during the same part of the gestational period. However, it would be problematical to interpret these findings in terms of possible RFR teratogenesis in humans, especially since high intensity levels of RFR were used. The results on tumor heating with RFR were not detailed enough to permit evaluation.

Lindauer, G. A., L. M. Liu, G. W. Skewes, and F. J. Rosenbaum
FURTHER EXPERIMENTS SEEKING EVIDENCE OF NONTHERMAL BIOLOGICAL EFFECTS OF MICROWAVE RADIATION
IEEE Trans. Microwave Theory Tech., Vol. 22, No. 8, pp. 790-793 (1974)

Study type: (3) Teratogenic and developmental abnormalities; IN VIVO; TENEBRIO MOLITOR (DARKLING BEETLE)

Effect type: Abnormalities in emergent adults due to RFR exposure of pupae

Frequency/wavelength: 9.0 GHz

Modulation: CW and Pulsed (0.25 microsec at 1.6 kHz and 16 Hz)

Power Densities: 20 mW Av. (CW and pulsed) equiv. to 17.1 mW/cm.2

SAR: 20 mW Av equiv. to 41 mW/g for large pupae (157 mg)

Exposure conditions: 4 pupae concurrently in individual terminated waveguide, with body along waveguide axis and head toward source. Exposure durations were 2 hr at 20 mW and 4 hr at 10 mW. Some pupae were aligned with long axis parallel to electric vector.

AUTHOR ABSTRACT: Carpenter and Livstone's experiments on beetle pupae are repeated and extended. In the experiments conducted, increased incidence of abnormal development occurred due to exposure to microwave energy, both CW and pulsed. This effect was observed at the power level of 8.6 mW/cm.2. Measurements are reported which specify the microwave environment encountered by the insect.

OTHER INFORMATION: Pupae in the first or second day of pupation, harvested from larvae raised on Special K and occasional pieces of potato or apple for moisture, were exposed individually in waveguide with heads toward the source in groups of four to 20 mW at 9.0 GHz CW, or to 0.25-microsecond pulses either at a peak power of 50 W and 1600 pps or 5 kW and 16 pps corresponding to an average power of 20 mW and a power density of 17.1 mW/cm.2 on the waveguide axis. Exposures were for 20 min. Ambient temperature was 21 deg C. Temperature rise during RFR exposure, measured with a thermocouple inserted in the abdomen, was 1.76 deg C (for head toward source), attained during first 8 min of exposure. Other groups were exposed to 10 mW (8.6 mW/cm.2) for either 2 or 4 hrs. Some pupae were exposed to 20 mW for 2 hrs with their long axes

parallel to the electric field. A group of pupae in their fifth day of pupation was exposed, with heads toward the source, to 20 mW for 2 hrs. Control groups consisted of sham-exposed pupae, those placed in an oven at 29 deg C (8 deg above ambient) for 2 hr, and those permitted to complete pupation without treatment. Following emergence, the adult beetles were categorized as N (normal), G (abnormal, with 3 subcategories), as used by Carpenter and Livstone (1971). Statistical comparisons of the control groups showed no significant differences at the 5% level. For the combined control groups (375 specimens), 240 (64%) were normal, 77 (21%) were dead, and 58 (15%) were abnormal. For those exposed to 20 mW for 2 hrs (185), 44 (24%) were normal, 40 (22%) were dead, and 101 (55%) were abnormal, with the percentages of normal and abnormal beetles being significantly different from controls. Significant differences were also found between the other RFR-exposed and control groups. Comparisons of results for those irradiated at 20 mW CW for 2 hrs with those exposed under the other RFR conditions used showed significant differences only for the group of 61 pupae irradiated at 10 mW for 2 hrs: 18 (30%) normal, 19 (31%) dead, and 24 (39%) abnormal. For the latter exposure regimen, the percentage of normal beetles was higher and the percentage of abnormal beetles was lower than for those irradiated at 20 mW, as would be expected, but the percentage of dead specimens was higher. The results for 82 pupae irradiated at 10 mW for 4 hrs were: 20 (24.4%) normal, 24 (29.3%) dead, and 38 (46.3%) abnormal. Measurements of the complex dielectric constant of homogenized pupal tissue are also discussed. Measurements of the voltage standing wave ratio indicated that the electric field varies considerably with distance along the length of a waveguide-mounted pupa and is highest at its center.

FINAL CRITIQUE: Carpenter and Livstone (1971) found a teratogenic effect of CW RFR on pupae for waveguide exposures to 40 mW for 2 hrs. The results of Lindauer et al. (1974) essentially confirm the findings of Carpenter and Livstone with CW for waveguide powers of 10 and 20 mW and also show no significant differences between results for pupae exposed to CW and those exposed to either type of pulsed RFR. However, the data do not reveal any clear dependence of the effect on dose rate or total dose, and are otherwise rather ambiguous. Based on the findings of Pickard and Olsen (1979), it is likely that the presence of uncontrolled non-RFR factors contributed to the numerical results. The measurements of complex dielectric constant for homogenized pupal tissue by Lindauer et al. (1974) are of doubtful utility in this context because of the nonuniform distribution of water in intact pupae and the spatial variation of fields along the length of a pupa.

REFERENCES: Carpenter, R. L. and E. M. Livstone, EVIDENCE FOR NONTHERMAL EFFECTS OF MICROWAVE RADIATION: ABNORMAL DEVELOPMENT OF IRRADIATED INSECT PUPAE, IEEE Trans. Microwave Theory Tech., Vol. 19, No. 2, pp. 173-178 (1971)

0048-6604/79/1112-S027, Pickard, W. F. and R. G. Olsen, DEVELOPMENTAL EFFECTS OF MICROWAVES ON TENEBRIO: INFLUENCES OF CULTURING PROTOCOL AND OF CARRIER FREQUENCY, Radio Sci., Vol. 14, No. 6S, pp. 181-185 (1979)

Liu, L. M., F. J. Rosenbaum, and W. F. Pickard
THE RELATION OF TERATOGENESIS IN TENEBRIO MOLITOR TO THE INCIDENCE OF LOW-LEVEL MICROWAVES
IEEE Trans. Microwave Theory Tech., Vol. 23, No. 11, pp. 929-931 (1975)

Study type: (3) Teratogenic and developmental abnormalities; IN VIVO; TENEBRIO MOLITOR (DARKLING BEETLE)

Effect type: Abnormalities in emergent adult beetles due to RFR exposure of pupae; dose-response reciprocity

Frequency/wavelength: 9.0 GHz

Modulation: CW

Power Densities: 0.05 to 20 mW or 0.043 to 17 mW/cm.2

SAR: About 40 mW/g for 20 mW

Exposure conditions: 4 pupae concurrently in individual terminated waveguides, with body along waveguide axis and head toward source. Exposures were at 0.05 to 20 mW for 2 hrs, or at 0.5 to 16 mW for durations of 8 to 0.25 hrs corresponding to 4 mW-hr

AUTHOR ABSTRACT: The teratogenic effects of irradiation by low-level microwaves have been studied using the pupae of the darkling beetle Tenebrio molitor. For exposures of 2-h duration, statistically significant increases in teratogenesis were observed at waveguide power levels down to 0.2 mW; the pupation time increased monotonically with the power. Exposures of various durations and powers at a constant dosage of 9 mW/h strongly suggested that it is the total dosage which determines the level of teratological damage.

OTHER INFORMATION: Groups of four one-to-two day pupae harvested from larvae raised on Special K were exposed individually to 9 GHz CW in waveguide, with heads toward the source, for 2 hrs at 0.05, 0.1, 0.2, 1, 2, 10, or 20 mW. Other groups were exposed for 8 hrs at 0.5 mW, 4 hrs at 1 mW, 2 hrs at 2 mW, 1 hr at 4 mW, 0.5 hr at 8 mW, or 0.25 hr at 16 mW, corresponding to 4 mW-hr (not 4 mW/hr as stated in the paper). Control pupae were sham-irradiated. Pupation of RFR-exposed and control pupae was in a darkened environmental chamber at 21 deg C. Emergent adult beetles were categorized as N (normal), D (died during pupation), or G (abnormal, with 3 subcategories) in the manner of Carpenter and Livstone (1971). Out of the control group of 298 for the first set of exposures, 208 (70%)

were normal, 54 (18%) were dead, and 36 (12%) were abnormal; approximately the same percentages were obtained for the 159 specimens in the second control group. Regression analyses of percentages of each category versus the logarithm of the power for the 2-hr exposures yielded straight lines having negative slope for category N and positive slopes for categories D and G, with correlation coefficients of -0.99, 0.99, and 0.97, respectively. These results indicate the existence of a threshold between 0.05 and 0.1 mW (or between 0.1 and 0.2 mW-hr). However, the differences between the groups exposed to 2 and 1 mW were not statistically significant at the 5% level, and similarly for the groups exposed to 0.1 and 0.05 mW. Pupation duration was found to be increased by RFR exposure, confirming that finding of Carpenter and Livstone (1971); specifically, a regression line for pupation duration versus the logarithm of the power had a positive slope (0.99 correlation coefficient) starting at about 0.05 mW. The results of the test for reciprocity at 4 mW-hr indicated no significant differences among the various power-duration products used, indicating that the total energy deposited (total dosage) determines the extent of teratologic effect.

FINAL CRITIQUE: The results of this investigation confirm and extend the findings of Carpenter and Livstone (1971) and Lindauer et al. (1974) that RFR can be teratogenic to Tenebrio molitor. Although reciprocity between exposure duration and incident power was demonstrated for 4 mW-hr and may be true for other values of total incident energy, the decrease in the percentage of normal beetles for constant exposure duration (2 hrs) is not linearly dependent on the incident power but on its logarithm. This point, as well as the existence of thresholds, indicates that the effect is not solely dependent on total dose. It is also noted that in the light of the large spatial variation of SAR in pupae measured by Olsen and Hammer (1978) and the nonuniform distribution of water in pupae, the hypothesis proposed by Carpenter and Livstone (1971) and reiterated by Liu et al. (1975) that such teratogenic effects are nonthermal remains unproved.

REFERENCES: Carpenter, R. L. and E. M. Livstone, EVIDENCE FOR NONTHERMAL EFFECTS OF MICROWAVE RADIATION: ABNORMAL DEVELOPMENT OF IRRADIATED INSECT PUPAE, IEEE Trans. Microwave Theory Tech., Vol. 19, No. 2, pp. 173-178 (1971)

Lindauer, G. A., L. M. Liu, G. W. Skewes, and F. J. Rosenbaum, FURTHER EXPERIMENTS SEEKING EVIDENCE OF NONTHERMAL BIOLOGICAL EFFECTS OF MICROWAVE RADIATION, IEEE Trans. Microwave Theory Tech., Vol. 22, No. 8, pp. 790-793 (1974)

Olsen, R. G. and W. C. Hammer, THERMOGRAPHIC ANALYSIS OF WAVEGUIDE-IRRADIATED INSECT PUPAE, in Abstracts of Open Symposium on the Biological Effects of Electromagnetic Waves, Helsinki, Finland, p. 62 (1978)

McRee, D. I. and P. E. Hamrick
EXPOSURE OF JAPANESE QUAIL EMBRYOS TO 2.45-GHZ MICROWAVE RADIATION DURING DEVELOPMENT
Radiat. Res., Vol. 71, No. 2, pp. 355-366 (1977)

Study type: (3) Teratogenic and developmental abnormalities, (8) Immunological, (9) Biochemical/physiological; IN VIVO; JAPANESE QUAIL

Effect type: RFR induced deformities, weight changes of body and organs, changes in hematological parameters

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 5 mW/cm.2

SAR: 4.03 W/kg

Exposure conditions: 6X5 arrays of eggs in far field at constant temperature and humidity. Each array was exposed 24 hrs/day for the first 12 days of development. Arrays were automatically turned 90 deg every 2 hrs. Control arrays were sham-exposed but otherwise treated similarly.

AUTHOR ABSTRACT: Japanese quail embryos were exposed during the first 12 days of development to 2.45 GHz microwave radiation at an incident power density of 5 mW/cm.2 and specific absorption rate of 4.03 mW/g. No gross deformities were observed in the exposed quail when examined and sacrificed at 24-36 hr after hatch. No significant changes in the total body weight or weights of the heart, liver, gizzard, adrenals, and pancreas were found in the treated birds. Hematological parameters were also measured in the study. The results showed a statistically significant increase in hemoglobin and statistically significant decrease in monocytes in birds treated with microwave radiation. No statistically significant changes in hematocrits, red blood cells, total white blood cells, lymphocytes, heterophils, basophils, or eosinophils were detected.

OTHER INFORMATION: The authors stated that each array was exposed with the major axes of the eggs parallel to the electric field (vertical). However, they also stated that each array was automatically turned 90 deg every 2 hrs, implying that the eggs were exposed with their major axes parallel to the magnetic field (horizontal) for half the time. Temperature distributions along the central major axis of one of the eggs

in an array (with major axes vertical) and along the minor axis parallel to the propagation direction (front-to-rear) were measured with a thermistor shown to be nonperturbing. The results showed approximately constant temperature along the vertical axis, but the center was found to be 0.4 deg C hotter than the front and 0.6 deg C hotter than the rear. (Analogous measurements for horizontal exposure were not presented.) Temperatures of the eggs in an array exposed to RFR with the chamber at 35.5 deg C ranged from 37.5 to 38.0 deg C. Three arrays of 30 eggs each were exposed for the first 12 days (24 hrs/day) of development and transferred to a normal hatching incubator for the remainder of the 16- to 17-day incubation period. Control arrays were sham-irradiated and incubated for the same periods. The neonatal quail were weighed and visually examined between 24 and 36 hrs after hatching for deformed feet, toes, and beak, and for microphthalmia or exencephalia. They were then euthanized, blood samples were obtained, and the internal organs were examined and weighed. The first array was exposed to RFR with the chamber at 37 deg C (the optimum incubation temperature). The temperatures of the eggs stabilized at between 39.5 and 40 deg C, so the chamber used for sham irradiation was set at 40 deg C. Two of the RFR-exposed and none of the control eggs hatched, with deformities evident in both groups of embryos. These results were ascribed to hyperthermia in both exposed and control eggs. The second array was exposed with the chamber at 35.5 deg C. The equilibrium temperatures reached were between 37.5 and 38.0 deg C, so the control chamber was set at the latter value. A larger percentage of the exposed than the control eggs hatched (numbers not explicitly stated). No gross deformities of organs or other structures were detected for either group and no statistically significant differences were found in body or organ weights or in hematocrit, total white blood cells, red blood cells, lymphocytes, heterophils, basophils, or eosinophils. The only significant differences were in higher mean hemoglobin (about 4%) and lower mean monocyte count (31%) for the RFR-exposed group. The authors speculate that the lower monocyte count could reduce the defense of such birds against certain types of infection. Regarding the third group, even though the authors initially state that three groups were exposed to RFR, the text implies that the third group was incubated at 37.5 deg C without either RFR or sham exposure. No statistically significant differences between this control group and the second sham-exposed group were found.

FINAL CRITIQUE: It is noted that the mean internal distributions of temperature within the RFR-exposed group are unknown if the eggs were exposed half the time with their major axes horizontal, but are likely to be smaller than the values

cited for vertical exposure. However, the differences of mean temperature from egg to egg in the arrays are relatively large (up to 0.5 deg C), rendering it difficult to associate either of the two positive findings (elevated hemoglobin and lowered monocyte count) specifically with RFR exposure. Regarding the authors' speculation that the latter effect could reduce the immunocompetence of such birds, see Hamrick et al. (1977).

REFERENCES: Hamrick, P. E., D. I. McRee, P. Thaxton, and C. R. Parkhurst, HUMORAL IMMUNITY OF JAPANESE QUAIL SUBJECTED TO MICROWAVE RADIATION DURING EMBRYOGENY, Health Phys., Vol. 33, pp. 23-33 (1977)

Olsen, R. G. and W. C. Hammer
THERMOGRAPHIC ANALYSIS OF WAVEGUIDE-IRRADIATED INSECT PUPAE
in Abstracts of Open Symposium on the Biological Effects of Electromagnetic Waves, Helsinki, Finland, p. 62 (1978)

Study type: (3) Teratogenic and developmental abnormalities, (16) Physical methods/dosimetry; IN VITRO; TENEBRIO MOLITOR (DARKLING BEETLE)

Effect type: Spatial distribution of SAR in pupa exposed to RFR in waveguide by thermographic imaging

Frequency/wavelength: 1.3, 5.95, and 10 GHz

Modulation: CW

Power Densities: Not stated

SAR: See results

Exposure conditions: Pupa in waveguide at three orthogonal orientations

AUTHOR ABSTRACT: Pupae of the insect TENEBRIO MOLITOR were thermographically imaged during waveguide irradiation to determine the magnitude and spatial distribution of the absorbed radiation. TENEBRIO pupae have been studied in regards to microwave-induced teratogenesis for some time, but up to now the small size of the insect has prevented a detailed dosimetry. A high-resolution thermographic imaging system was used to obtain a real-time sequence of thermograms showing the evolution of heat in waveguide-mounted insects absorbing microwave energy. From these data both local and overall average specific absorption rate (SAR) were calculated. Three frequencies were used to show the general features of resonance absorption: L-band (1.3 GHz), C-band (5.95 GHz), and X-band (10 GHz); and three orthogonal orientations were used except at X-band where the height of the waveguide prevented electric field polarization. Longitudinal slots were cut in the broad wall of the waveguide through which the pupae were imaged. It is shown that our results compare favorably with previous theoretical and experimental work at X-band, and at this frequency the ratio of peak-to-average SAR for the longitudinal orientation is about 2.0 with a maximum located near the abdomen-thorax interface.

INITIAL CRITIQUE: The large spatial variations of local SAR in waveguide-exposed Tenebrio pupae reported in this abstract are significant because such variations would not be obtained in

pupae heated (without RFR) in an oven or other uniform-temperature environment. Since non-RFR heating was used by Carpenter and Livstone (1971) and subsequent investigators to distinguish between thermal and nonthermal RFR effects, the hypothesis that the RFR teratogenic effects found by them are nonthermal is negated. (A more detailed, final analysis of this investigation by Olsen and Hammer is planned when a copy of the full paper is obtained.)

REFERENCES: Carpenter, R. L. and E. M. Livstone, EVIDENCE FOR NONTHERMAL EFFECTS OF MICROWAVE RADIATION: ABNORMAL DEVELOPMENT OF IRRADIATED INSECT PUPAE, IEEE Trans. Microwave Theory Tech., Vol. 19, No. 2, pp. 173-178 (1971)

Pickard, W. F. and R. G. Olsen
DEVELOPMENTAL EFFECTS OF MICROWAVES ON TENEBRIO: INFLUENCES OF CULTURING PROTOCOL AND OF CARRIER FREQUENCY
0048-6604/79/1112-S027, Radio Sci., Vol. 14, No. 6S, pp. 181-185 (1979)

Study type: (3) Teratogenic and developmental abnormalities; IN VIVO; TENEBRIO MOLITOR (DARKLING BEETLE)

Effect type: Abnormalities in emergent adult beetles due to RFR exposure of pupae as influenced by culturing aspects

Frequency/wavelength: Approx 6 and 10 GHz

Modulation: CW

Power Densities: 91 V/m. 1.53 A/m, or 110 W/m.2 at 6 GHz; 50 W/m.2 at 10 GHz

SAR: 130, 54, 130 W/kg respectively at 6 GHz; 45 W/kg at 10 GHz

Exposure conditions: Far-field, in arrays of 3X4 pupae; standing-wave exposures obtained by use of reflection plate, with arrays exposed in max E-field and H-field planes for 2 hrs at 6 GHz; traveling-wave exposures for 13 hrs at 6 GHz and 4 hrs at 10 GHz, obtained by omitting reflection plate.

AUTHOR ABSTRACT: First-day pupae of the darkling beetle Tenebrio molitor were exposed to CW microwaves in the far field of a horn-irradiated, temperature-controlled, anechoic chamber. They and controls were allowed to develop to the adult stage and were then examined for the presence of gross morphological abnormalities. Control pupae from an in-house colony developed significantly fewer developmental abnormalities than control pupae purchased from an outside supplier. The following experiments were performed at 5.95 GHz: (1) Pupae were held parallel to the electric-field vector for two hours at an E-field maximum (91 V/m RMS) of a standing-wave distribution; at a nominal dose rate of 130 W/kg, no effect was observed in either sample of pupae. (2) Pupae were held parallel to the magnetic-field vector for two hours at an H-field maximum (1.53 A/m RMS) of a standing-wave distribution; at a nominal dose rate of 54 W/kg, no effect was seen in colony pupae but significant effects were observed in cultures of other pupae. (3) Pupae were held parallel to the electric-field vector in a traveling-wave distribution at 110 W/m.2; only outside pupae were used, but no effect was detected for 13-hour exposures. Another experiment was performed at 10.025 GHz: Pupae were

held antiparallel to Poynting's vector for four hours in a traveling-wave field at 50 W/m.2; no effect was detected in colony pupae but a marginally significant effect was observed in the outside pupae. It was also demonstrated in samples of both pupal types that the condition under which they were maintained during development within the pupal stage could significantly affect the incidence of abnormalities in the adults.

OTHER INFORMATION: Pupae from two sources were used for RFR exposure and controls in each experiment. "Colony pupae" were from a colony derived initially as larvae from one supplier and maintained at the authors' laboratory on ground Purina dairy meal and sliced potatoes. "K-pupae" were purchased as larvae from another supplier in 3 batches and maintained on Kellogg's Special K and sliced potatoes. Ambient temperature was 20-25 deg C and relative humidity about 70%. Day-one pupae having no externally discernible abnormalities with a dissection microscope were used. Each was inserted into a perforated No. 0 gelatin capsule for exposure. For standing-wave exposures (using a reflection plate), one 3X4 array was mounted in a plane of maximum E with the long axes of the pupae parallel to E; another array was mounted in a plane of maximum H (a quarter wavelength from the first array) with the long axes parallel to H. For the "parallel" traveling-wave exposures (without reflection plate), the long axes were parallel to the E vector (vertical), and for the "perpendicular" traveling-wave exposures, the long axes were parallel to the propagation vector, with the heads toward the source. Approximately equal numbers of pupae (in gelatin capsules) for each exposure were placed in a darkened container at 23-25 deg C as controls. Control and exposed pupae were allowed to develop in the dark in perforated roughened polyethylene centrifuge vials. After emergence, adult beetles were categorized as "N" for normal, "D" for dead, and "G" for anomalous, with G divided into various subcategories, and the numbers of each type for each situation were treated statistically. The results for max E-plane exposure (at 5.95 GHz) of colony-pupae and of K-pupae from the first batch of larvae delivered showed no statistically significant differences between RFR-exposed and controls groups for either colony-pupae or K-pupae. However, the numbers of non-normal (D+G) beetles from the control K-pupae were significantly larger than from the control colony-pupae. Also, the results for max H-plane exposure showed a statistically significant effect of RFR on the K-pupae but not on the colony-pupae, with the differences between the two control populations again being significant. To test this apparent RFR susceptibility of K-pupae, the max H-plane experiment was repeated with K-pupae

from each of the three batches of larvae received. A doubtfully significant deleterious RFR effect was obtained for the first batch, no significant effect was found for the second batch, and a significant beneficial effect (fewer non-normal beetles) of RFR was seen for the third batch. Ambiguous results were also obtained for pupae exposed in the "parallel" and "perpendicular" traveling-wave configurations (for 13 hrs at 5.95 GHz and 4 hrs at 10.025 GHz, respectively). Comparisons of control pupae developed in roughened polyethylene centrifuge tubes with those developed in smooth polystyrene cups (used by other investigators, e.g., Green et al., 1979) indicated that use of the latter is deleterious to both colony-pupae and K-pupae. A sample of control K-pupae from the first batch (which was maintained longest) yielded a marginally significant higher number of non-normal beetles than a sample developed concurrently from the second batch. The authors conclude that despite the inconsistencies, the results point toward the existence of RFR teratogenesis.

FINAL CRITIQUE: Although some of the findings of these investigators were ambiguous, signifying the presence of residual uncontrolled factors, their results indicate that much of the variability in the results of other investigators, notably those of Carpenter and Livstone (1971), Lindauer et al. (1974), Liu et al. (1975), and Green et al. (1979, 1977), may be due to uncontrolled differences in source larvae, pupae maintenance regimes and handling protocols, pupae containers used for pupation, and ambient temperature. Pickard and Olsen conducted their investigations in ambient relative humidicies of about 70%. Green et al. (1979) found that pupae cultured and exposed at ambient relative humidities less than 35% appeared to be more susceptible to RFR teratogenesis than pupae similarly treated at higher ambient humidities, indicating the possible importance of this factor. Despite the large variations among the data obtained by all these investigators, their findings indicate that RFR at the frequencies, dose rates, and exposure durations used can be teratogenic to Tenebrio molitor pupae. However, Olsen and Hammer (1978), using thermographic imaging, found large spatial variations of SAR in pupae irradiated in waveguide at power densities comparable to those used in the investigations cited above; such spatial temperature distributions would not be obtained by heating pupae in a uniform temperature environment. Thus, such RFR teratogenesis may be thermally induced, in contradistinction to the nonthermal hypothesis of Carpenter and Livstone (1971) and later investigators.

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Rugh, R., F. I. Ginns, H. S. Ho, and W. M. Leach
RESPONSES OF THE MOUSE TO MICROWAVE RADIATION DURING ESTROUS CYCLE AND PREGNANCY
Radiat. Res., Vol. 62, pp. 225-241 (1975)

Study type: (3) Teratogenic and developmental abnormalities, (7) Endocrinological, (9) Biochemical/physiological; IN VIVO; MOUSE

Effect type: Higher RFR sensitivity (lethality) during estrus; RFR-induced fetal anomalies and hematoma

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 116 to 138 mW/cm.2

SAR: About 5 to 8 cal/g (about 123 mW/cm.2 for 2 to 5 min)

Exposure conditions: Individually in waveguide at constant forward power for specified durations (2 to 5 min) at 25 deg C, 50% relative humidity, and 38 l/min airflow

AUTHOR ABSTRACT: A new facility for microwave irradiation of mice which will provide reproducible dosimetry is described. The waveguide used provided the integral dose rate to experimental animals under stable and controlled environmental conditions of relative humidity and temperature, variables which have been found to be critical in microwave studies. In terms of average absorbed lethal dose, the female mouse was found to be more sensitive to microwave irradiation during estrus than during diestrus. Teratogenesis (e.g., exencephalies) after sublethal irradiation of pregnant mice at 8 gestation days resulted from absorbed doses within the range of 3-8 calories per gram of body weight, and was never an all-or-none response. The incidence and variety of effects produced (hemorrhage, resorption, stunting, and fetal death) indicate that the cause and effect relationships are neither linear nor well enough established and understood to permit prediction of the biological effects either in the mouse or other species. As the absorbed dose of radiant energy is increased to the 8-day pregnant mouse, the probability of it producing at least one exencephaly is likewise increased. Nevertheless, the determination of the absorbed dose of microwave energy in each mouse is one step closer to determining the precise absorbed-dose-effect relationship for microwave exposures. A total of 1096 mice were exposed to

microwave radiation and separately monitored to gather the related data.

OTHER INFORMATION: The forward, reflected, and transmitted power were integrated over the exposure periods, from which integral doses for the whole animal (in cal.) and average doses per unit mass (D/M in cal/g) were calculated. Time-variations of reflected power were used as an indication of animal movements; cessation of such variations signified death. In a lethality study with 77 2-month-old male mice, the lethal average dose per unit mass (LD/M) was found to decrease approximately linearly with the temperature-humidity index (THI) derived by Mumford (1969). At a THI of about 70 (23.5 deg C and 50% humidity), the value of LD/M for a group of 90 female mice in estrus, exposed to about 138 mW/cm.2, was found to be 10.65 cal/g, whereas the value for another group of 74 mice in diestrus (having approximately the same mean weight) was 11.50 cal/g. The difference was statistically significant (at less than the 1% level), and was tentatively ascribed to the somewhat higher (about 6%) tissue hydration during estrus or possibly to changes in hormone-dependent sensitivity to RFR. For the anomalous-fetus studies, pregnant mice on day 8 of gestation were exposed to 123 mW/cm.2 for durations up to 5 min, corresponding to sublethal values of D/M ranging from 3 to 8 cal/g. On gestational day 18, the litters were examined for resorptions, and for dead, stunted, malformed, and apparently normal fetuses. A plot of the percentage of normal fetuses in each litter versus the value of D/M showed a considerable number of litters (too dense to count) with 100% normal fetuses (over the exposure range from 3.4 to 7.8 cal/g), 6 litters with no normal fetuses (over the range from 5.8 to 7.7 cal/g), and the remainder with various intermediate percentages. A plot of the percentage of resorptions per litter (the authors' Fig. 7) showed many with none (up to 7.7 cal/g), 3 with 100% (all above 6 cal/g), and the remainder with intermediate values. The incidence of exencephaly (brain hernia) was also similarly plotted (Fig. 8). Many litters showed none (spanning the entire dose range). The maximum was 60% (2 litters at about 7 cal/g), and the average expectancy at 8 cal/g was only 12%. The authors divided the dose range from 2.6 to 8.6 cal/g into 3 equal intervals and tabulated the percentages of litters in each interval having at least one exencephaly. The results show that the percentage of litters having this anomaly increases with dose. To distinguish between such abnormalities and the immediate effects of RFR, the authors discuss 2 fetuses removed immediately after exposure, on gestational day 15, at 123 mW/cm.2 for less than 5 min. The predominant effect was the formation of hematomas. The paper by Rugh et al. (1974) contains essentially the same information as this paper.

FINAL CRITIQUE: Apparently no controls were used, presumably under the assumption that the natural incidence of exencephaly is relatively rare. In a similar study by Chernovetz et al. (1975), who used C3H/HeJ mice instead of the CF1 strain, about 20% of the fetuses from their control dams were abnormal. The incident power densities (about 116 to 138 mW/cm.2) and durations (2 to 5 min) used by Rugh et al. are well within the acute thermal range (up to and including lethality). For reasons that are not understood by us, the authors state that "...there seems to be no evidence of a threshold effect within the range of this study. It should be pointed out that data were not obtained below doses of about 2.5-3.0 cal/g, since preliminary studies at these lower radiation levels showed no teratogenesis." These two statements are contradictory, i.e., the second one implies the existence of a threshold. In fact, the data points in the authors' Fig. 8 were used by us to determine the numbers of exencephalic fetuses in dose intervals of 0.5 cal/g over the range, assuming a mean of 10 fetuses per litter. Plotting the results showed the existence of a threshold of about 3.6 cal/g. A similar treatment of the points in Fig. 7 showed a threshold for resorptions at about 3.5 cal/g. Although these values may be in error because of the large scatter of the data, the existence of thresholds is evident. However, in view of the small ranges of exposure duration and dose rate used, it is not clear that these thresholds are really for integrated doses, but may represent dose-rate thresholds instead. The latter hypothesis is supported by the results of Berman et al. (1978), who exposed mice for 100 min (per day) at SARs from about 2 to 22 mW/g (3.4 to 28 mW/cm.2), which correspond to integrated doses from about 3 to 32 cal/g, with marginal teratogenic effects. In general, Rugh et al. conclude that teratogenic effects can be induced in mice at RFR levels that are not lethal to the dams, a finding that is at variance with that of Chernovetz et al. (1975), who concluded that near lethal levels of RFR were not teratogenic to mice. Among the possible reasons for these apparently contradictory results are the differences in exposure systems used by Chernovetz et al. and Rugh et al. (cavity versus waveguide), the use of multiple versus individual animal exposures, gross uncertainties in actual doses, the mouse-strain difference, differences in gestational day of treatment (day 11 through 14 versus day 8), handlng of the dams, and the aforementioned lack of controls. Both groups of investigators indicate that caution is necessary in interpreting their findings with respect to possible RFR teratogenesis in humans, with which we concur.

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Stavinoha, W. B., A. Modak, M. A. Medina, and A. E. Gass
GROWTH AND DEVELOPMENT OF NEONATAL MICE EXPOSED TO HIGH-FREQUENCY ELECTROMAGNETIC FIELDS
USAF School of Aerospace Medicine, Brooks AFB, Texas; Final Report SAM-TR-75-51 on Contract F41609-74-C-0018, submitted by University of Texas Health Science Center, San Antonio, Texas (1975)

Study type: (3) Teratogenic and developmental abnormalities; IN VIVO; MOUSE

Effect type: HF-RFR effects on growth and development of neonatal mice

Frequency/wavelength: 10.5, 19.27, or 26.6 MHz

Modulation: Pulsed

Power Densities: 5.8 kV/m in waveguide; 8.0 kV/m and 55 A/m in near-field synthesizer

SAR: Not measured

Exposure conditions: In the first study, 4-day-old mice were exposed for 20 min at 10.5, 19.27, or 26.6 MHz in a coaxial rectangular waveguide system. In the second study, 4-day-old mice were exposed at 19 MHz for 40 min/day on 5 consecutive days, in a near-field synthesizer.

AUTHOR ABSTRACT: Four-day-old mice were exposed to high-frequency electromagnetic radiation. Growth rate was followed for up to 16 weeks of age. No effect of irradiation on the growth and development of these neonatal mice was evident.

OTHER INFORMATION: In the first study, groups of 4-day-old Swiss Webster mouse pups were placed in plastic containers on the center conductor of a coaxial rectangular waveguide terminated with a 50-ohm dummy load and exposed once for 20 min at 10.5, 19.27, or 26.6 MHz. The electric field, 5.8 kV/m, was measured with a monopole. Control groups were kept in similar containers outside the exposure chamber. The mice were weighed daily for the next 21 days. Graphs of weight versus age for the three frequencies show virtually no differences between exposed and control animals. In the second study, litters of 4-day-old pups from 20 female mice were divided into three groups: (1) Control pups; (2) Thermal-control pups, held at 37 deg C for 40 min/day on 5 consecutive days; and (3) Irradiated pups, exposed at 19 MHz for 40 min/day on 5

consecutive days in a near-field synthesizer with the electric field (8 kV/m) and the magnetic field (55 A/m) in coincident planes. The pups were weighed daily before each treatment and until they were 21 days old, at which time the males and females were separated. Subsequently the mice were weighed weekly for a total of 16 weeks. Statistical analyses of growth curves showed no significant differences among the three groups either for the males or the females. The final weights of the males were somewhat higher than those of the females.

FINAL CRITIQUE: The fields used in these studies were very intense, yet they did not affect mouse growth. The investigators point out that the sizes of the mice were much smaller than the wavelengths used, so that relatively little energy was absorbed. (It would have been interesting to determine SARs.) For this reason, it would be inappropriate to apply these negative findings to humans exposed at frequencies in the same range.

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In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 261-267 (1974) (See "Epidemiologic" for analysis.)

[2]Stern, S., L. Margolin, B. Weiss, S.-T. Lu, and S. M. Michaelson
MICROWAVES: EFFECT ON THERMOREGULATORY BEHAVIOR IN RATS
0036-8075/79/1207-1198, Science, Vol. 206, pp. 1198-1201 (7 Dec 1979)

[2]Thomas, J. R., J. Schrot, and R. A. Banvard
BEHAVIORAL EFFECTS OF CHLORPROMAZINE AND DIAZEPAM COMBINED WITH LOW-LEVEL MICROWAVES
0191-3581/80/020131, Neurobehav. Toxicol., Vol. 2, pp. 131-135 (1980)

[2]Thomas, J. R., L. S. Burch, and S. S. Yeandle
MICROWAVE RADIATION AND CHLORDIAZEPOXIDE: SYNERGISTIC EFFECTS ON FIXED-INTERVAL BEHAVIOR
0036-8075/79/0330-1357, Science, Vol. 203, pp. 1357-1358 (1979)

Analyses under

(6) BEHAVIORAL

Adair, E. R. and B. W. Adams
MICROWAVES MODIFY THERMOREGULATORY BEHAVIOR IN SQUIRREL MONKEY
0197-8462/80/0102-0101, Bioelectromagnetics, Vol. 1, No. 1, pp. 1-20 (1980)

Study type: (6) Behavioral; IN-VIVO; SAIMIRI SCIUREUS
Effect type: Behavioral thermoregulation changes under microwave and infrared irradiation

Frequency/wavelength: 2,450 MHz

Modulation: CW

Power Densities: Range from 1 to 22 mW/cm.2

SAR: 0.15 to 3.25 W/kg

Exposure conditions: Unanesthetized squirrel monkeys chair-restrained in far-field (1.85 m from front edge) of 15 dB standard gain horn in 1.83 m X 1.83 m X 2.45 m anechoic chamber. Chamber temperature regulated by monkey responses during 10-min exposure to approx 34-36 deg C.

AUTHOR ABSTRACT: Squirrel monkeys (Saimiri sciureus) trained to regulate environmental temperature (Ta) behaviorally were exposed in the far field of a horn antenna to ten-minute periods of 2,450 MHz CW microwaves. Incident power density ranged from 1 to 22 mW/cm.2. The corresponding specific absorption rate (SAR), derived from temperature increments in saline-filled styrofoam models, ranged from 0.15 to 3.25 W/kg. Controls included exposure to infrared radiation of equivalent incident energy and no radiation exposure. Normal thermoregulatory behavior produces tight control over environmental and body temperatures; most monkeys select a Ta of 34-36 deg C. Ten-minute exposures to 2,450 MHz CW microwaves at an incident power density of 6-8 mW/cm.2 stimulated all animals to select a lower Ta. This threshold energy represents a whole-body SAR of 1.1 W/kg, about 20% of the resting metabolic rate of the monkey. Thermoregulatory behavior was highly efficient, and skin and rectal temperatures remained stable, even at 22 mW/cm.2 where the preferred Ta was lowered by as much as 4 deg C. No comparable reduction in selected Ta below control levels occurred during exposure to infrared radiation of equal incident power density.

OTHER INFORMATION: Test subjects were 3 adult male squirrel monkeys (5 to 7 yrs of age, 750 to 1,100 g) that had been highly trained to regulate environmental temperature by adjusting air flows of different temperature into the anechoic

test chamber. The animals were exposed individually. Each was restrained in a lucite chair in the far-field of a 15-dB standard gain horn. A valve system allowed air from one or the other of two temperature-regulated sources to circulate through a restricted volume of the test chamber where the animal was located. The preset temperatures of these air sources were 15 and 55 deg C. The animal was initially exposed to airflow at one of these temperatures. By pulling a response cord, the animal could cause the airflow to change to the second temperature for 15 sec. Additional responses in this period were without consequence. At the end of 15 sec the airflow reverted to the initial temperature until the next response cord activity by the monkey. The ambient temperature, Ta, was monitored by a thermocouple in the chamber outlet. Under long-term exposure, all animals responded at a frequency that produced an average Ta of 35-36 deg C. Thermocouples were also used (with appropriate precautions to minimize RFR pickup) to measure rectal and weighted mean skin temperature (a combination of foot, leg, abdomen and tail skin temperature) under some exposure conditions. All experimental sessions were conducted in the presence of a 73-dB SPL masking noise to guard against auditory cues to the presence or absence of RFR. Calibration of the RFR exposure field was carried out using a Narda Model 8306B broadband isotropic radiation detector. An assessment of the whole-body energy absorption was based on measured temperature rises in 3 sizes of saline-filled cylindrical styrofoam models (0.75, 1.1, and 1.5 liter volumes). Specific absorption rate (SAR) was calculated to range from 0.5 W/kg at 5 mW/cm.2 to 5.8 W/kg at 40 mW/cm.2 for the 1.1 liter model. (Note that this is not a linear relationship, as would be expected.) In order to evaluate the contribution of skin heating alone (as compared with skin heating plus deeper tissue heating from RFR), the animals were exposed to infrared radiation (IR) at the same incident power density under identical exposure conditions. IR exposure was determined by a wide angle radiometer calibrated by a Bureau of Standards radiation lamp. The procedures for RFR (or IR) exposures were as follows: Five four-hour sessions of behavior thermoregulation were conducted on each animal to serve as baseline data. The animals selected their preferred air temperatures, as they had been trained to do. For the RFR exposure series, a two-hour stabilization period of behavioral thermoregulation was conducted. The animal was exposed to 10-min periods of 2,450-MHz CW RFR of increasing incident power density (1, 2, 4, 6, 8, and 10 mW/cm.2). Each 10-min RFR exposure was followed by a 10-min period of no RFR. The Ta was measured in the chamber air outlet. Three animals were tested, each for 5 experimental sessions. A further RFR exposure series was conducted on 2 animals at 8, 10, 12, 15, 18, and 22

mW/cm.2, again for 5 experimental sessions. IR exposures were carried out on 2 animals under identical conditions and incident power densities as those for the first RFR series. Results of the RFR exposures were as follows: During the first hr, the animal worked to stabilize the air temperature to a preferred 34-35 deg C (approximately). Mean skin and deep body temperature stabilized at approx 38 and 39.2 deg C, respectively. After 2 hrs, the ascending power intensity 10-min RFR exposures were carried out. The data were processed by computing mean values of all measured parameters for each animal. Grand means and standard errors were then computed across the 5 trials for each animal. Results of these computations show that for RFR exposure at 1, 2, and 4 mW/cm.2 there was no statistically significant change in Ta between the beginning and end of an exposure period. However, for 6 mW/cm.2 and above, RFR exposure caused a significant decrease in Ta over the exposure duration, as much as 4 deg C at 22 mW/cm.2. There was no such significant reduction for incident IR exposure. This was due in part to the animal reacting to higher IR power densities with frantic struggling and loss of thermoregulatory behavior, behavior not evidenced with RFR exposure. The authors interpreted the results to show that RFR triggered a thermal event occurring in one or more thermosensitive sites deep in the body tissue, and that the animals reacted to this trigger by adjusting their ambient temperature to maintain a preferred constant rectal and mean skin temperature.

FINAL CRITIQUE: This paper describes a well-conceived and competently conducted experiment. The animals performed a simple behavioral task very reliably, the paradigm was well established, and all aspects of the experiment were well thought out. Exposure conditions were calibrated accurately. There is a possible problem with the absorbed dosimetry. Thermocouples in the saline volume indicated significantly higher temperatures in the upper levels of the exposed phantoms. It is not indicated how temperature rise for the total volume was obtained, e.g., by shaking or otherwise. The mean SAR at which a significant decrease in Ta was observed during RFR exposure was calculated to be 1.1 W/kg. This is stated to be approximately 20% of the resting metabolic rate of the squirrel monkey.

Stern, S., L. Margolin, B. Weiss, S.-T. Lu, and S. M. Michaelson
MICROWAVES: EFFECT ON THERMOREGULATORY BEHAVIOR IN RATS
0036-8075/79/1207-1198, Science, Vol. 206, pp. 1198-1201 (7 Dec 1979)

Study type: (6) Behavioral; IN-VIVO; RAT

Effect type: Behavioral thermoregulation changes under microwave and infrared radiation

Frequency/wavelength: 2,450 MHz

Modulation: CW

Power Densities: 5 to 20 mW/cm.2

SAR: 0.20 W/kg per mW/cm.2

Exposure conditions: Unanesthetized male Long-Evans hooded rats (325 to 450 g) were individually trained to press a small lever to turn on an infrared (IR) lamp for 2 s. The rats were located in a small (20.4 by 20.8 by 40.5 cm) microwave anechoic box located in a refrigerated room. RFR at 5, 10, 15, or 20 mW/cm.2 was used to substitute for IR heat. Rats were exposed dorsally, with E-field parallel to long body axis.

AUTHOR ABSTRACT: Rats, with their fur clipped, pressed a lever to turn on an infrared lamp while in a cold chamber. When they were exposed to continuous-wave microwaves at 2450 megahertz for 15-minute periods, the rate at which they turned on the infrared lamp decreased as a function of the microwave power density, which ranged between 5 and 20 milliwatts per square centimeter. This result indicates that behaviorally significant levels of heating may occur at any exposure duration and intensities that do not produce measurable changes in many other behavioral measures or in colonic temperature. Further study of how microwaves affect thermoregulatory behavior may help us understand such phenomena as the reported "nonthermal" behavioral effects of microwaves.

OTHER INFORMATION: Six male Long-Evans hooded rats were individually trained to press a small lever in order to turn on an infrared lamp for 2 seconds. Additional responses during this 2-second period were without consequence. The fur of the rats was clipped. Test sessions involved placing a rat in a chamber located in a dark, refrigerated room. Control and exposure data were obtained with a room air temperature between 3.9 and 5.3 deg C. After initial training, the rat generally

pressed the lever at a nearly constant rate for several hours. This provided a baseline for studying the effects of 2.45-GHz CW RFR on thermoregulatory behavior. The results for each rat clearly indicated that the heat lamp was kept on for a certain fixed proportion of the time (different for each rat) without RFR; that RFR exposure decreased the number of responses turning on the heat lamp in a 15-min interval, and that this decrease was proportional to the power density of the RFR; that the presentation or removal of RFR caused an immediate change in the response rate; and that the change occurred even at the lowest power density tested (5 mW/cm.2).

FINAL CRITIQUE: Thermoregulatory behavior is known to be highly dependent on many factors, such as species, age, ambient temperature, airflow, humidity, circadian rhythm effects, drugs, etc. The present study, using rats, has been well-controlled for these and other environmental influences. For example, airflow in the vicinity of the lever was provided from above by a 100 cu ft per min blower. Airflow was measured as 6 m per min at the lever, using a hot-wire anemometer. Other environmental parameters were similarly carefully measured and reported. The authors concluded from their results that the rat responds to maintain a nearly constant thermal state. In the absence of RFR, the IR lamp is the sole heat source. When RFR is introduced, the rat compensates by reducing the response rate, thereby reducing the IR heat contribution. Thermoregulatory behavior therefore provides an index of the thermal burden contributed by RFR. The rapidity of this response is such that it cannot be mediated by deep-body (colonic) temperature rise, and indeed there is no change in colonic temperature at 5 mW/cm.2. The study provides interesting information on behavioral thermoregulation by RFR in rats that parallels a similar study in squirrel monkeys (Adair et al., 1980) where the threshold for RFR thermoregulation was 8 mW/cm.2.

REFERENCES: Adair, E. R., and B. W. Adams, MICROWAVES MODIFY THERMOREGULATORY BEHAVIOR IN SQUIRREL MONKEY, Bioelectromagnetics, Vol. 1, No. 1, pp. 1-20 (1980)

Thomas, J. R., J. Schrot, and R. A. Banvard
BEHAVIORAL EFFECTS OF CHLORPROMAZINE AND DIAZEPAM COMBINED WITH LOW-LEVEL MICROWAVES
0191-3581/80/020131, Neurobehav. Toxicol., Vol. 2, pp. 131-135 (1980)

Study type: (6) Behavioral, (18) Drug interactions; IN VIVO; RAT

Effect type: Modification of response-rate-altering effects of chlorpromazine and diazepam

Frequency/wavelength: 2.8 GHz

Modulation: Pulsed (2 microsec at 500 Hz)

Power Densities: 1 mW/cm.2 (average), 1 W/cm.2 (peak)

SAR: Approximately 0.2 mW/g

Exposure conditions: Rats restrained in a sleeve holder were exposed singly approximately 6.3 wavelengths (68 cm) in front of a standard-gain horn. E field was vertical. All exposures were in a microwave anechoic chamber 2.44 by 2.44 by 2.44 m. Ambient temperature was 21 + or - 1.5 deg C. Air flow at the position of the animal was 3 m/min.

AUTHOR ABSTRACT: Previous research findings on the interaction between drugs and microwave radiation were extended to chlorpromazine and to diazepam. The drugs were combined with a 1 mW/cm.2 pulsed microwave field (2.8 GHz) and effects were measured on a fixed interval (FI 1) schedule of food reinforcement with rats. Dose-effect functions with and without sham irradiation were established for each drug. At effective doses chlorpromazine consistently decreased rate of responding and reduced within-interval response patterning. Low to moderate doses of diazepam produced little change or increases in response rate, and higher doses produced decline in response rate. Response patterning within intervals was reduced by increasing doses of diazepam. The animals were exposed to the microwave field alone before test sessions combining the drugs with microwave radiation. Microwave exposure alone did not affect FI performance. Microwave radiation in combination with either drug did not produce any alterations in the dose-effect functions.

OTHER INFORMATION: Diazepam (also known as Valium) is a widely prescribed minor tranquilizer and skeletal muscle relaxant. Chlorpromazine is a phenothiazine derivative and in a different

class of drugs than diazepam and chlordiazepoxide which are benzodiazepine derivatives. Chlorpromazine is used as a sedative and as an antiemetic. Four male Long-Evans Hooded (LEH) rats and four male albino rats served as subjects. All were maintained at 80% of their free-feeding weights of 360 to 380 g throughout the study. They were trained on a fixed-interval (FI 1) reinforcement schedule for approximately 3 months until stable baseline performance was obtained. (This comprised a positively accelerated rate of responding in each FI period until a food pellet was obtained.) Dose-effect functions were then obtained for chlorpromazine over a dose range of 0.25 to 4 mg/kg with the LEH rats and for diazepam over a dose range of 0.5 to 20 mg/kg with the 4 albino rats. Drugs were administered i.p. 30 min before a session. Chlorpromazine lowered both response rates and index of curvature with increasing doses for all 4 animals. Response rates remained within baseline variability for doses out to approximately 1 mg/kg and declined thereafter. Diazepam showed little change or slight increases in response rates at doses up to approximately 2.5 mg/kg, with a decline in response rates thereafter. Index of curvature generally showed a decline with increasing doses of diazepam. Dose-effect functions for the two drugs were again obtained except that the animals were exposed to RFR at 1 mW/cm.2 (average) immediately after drug administration during the 30-min period before a session. The effects of chlorpromazine on FI performance did not seem to be greatly modified by the RFR exposure. The effects of diazepam on FI performance likewise did not seem to be greatly modified by the RFR exposure.

FINAL CRITIQUE: Although a clear-cut modification of similar FI performance under chlordiazepoxide had previously been obtained (Thomas et al., 1979) with similar RFR (2.45 GHz vs. 2.80 GHz in the present case, pulse parameters and average power densities identical), such modification was not obtained with chlorpromazine and diazepam in the present study. The authors postulate that the interaction of RFR and drugs is restricted to drugs that increase baseline response rates. However, one rat did exhibit a slight increase in response rate after doses of diazepam between 0.5 and 5 mg/kg. (The other 3 did not.) This rat did not show any difference between drug and drug-plus-RFR responses, weakening the postulate. Although diazepam and chlordiazepoxide are in the same class of drugs, this alone is not sufficient to predict synergistic or antagonistic effects with low-level RFR exposure.

REFERENCES: Thomas, J. R., L. S. Burch, and S. S. Yeandle, "Microwave Radiation and Chlordiazepoxide: Synergistic Effects on Fixed-Interval Behavior," Science, Vol. 203, pp. 1357-1358 (1979)

Thomas, J. R., L. S. Burch, and S. S. Yeandle
MICROWAVE RADIATION AND CHLORDIAZEPOXIDE: SYNERGISTIC EFFECTS ON FIXED-INTERVAL BEHAVIOR
0036-8075/79/0330-1357, Science, Vol. 203, pp. 1357-1358 (1979)

Study type: (6) Behavioral, (18) Drug interactions; IN-VIVO; RAT

Effect type: Potentiation of response-rate-increasing effects of chlordiazepoxide in fixed-interval (food reward) behavioral situation.

Frequency/wavelength: 2.45 GHz

Modulation: Pulsed; 2 microsec pulse width, 500 pulses per sec

Power Densities: 1 mW/cm.2 aver.

SAR: Not given

Exposure conditions: All exposures were conducted in a calibrated chamber lined with 20-dB microwave absorbing material. Exposures of animals suspended in a plastic mesh sleeve were in the near field, 3.75 wavelengths from the aperture of a standard gain horn. E field was vertical. Power density at the animal's head was 1 mW/cm.2. Ambient chamber temperature was 23 deg C.

AUTHOR ABSTRACT: In the presence of low-intensity pulsed microwave radiation, at an average power density of 1 milliwatt per square centimeter, the response-rate-increasing effects of chlordiazepoxide were potentiated in rats. The behavioral effects of a drug can be modified by brief exposure to a low-level microwave field even when the radiation level alone has no apparent effects on the behavior.

OTHER INFORMATION: Chlordiazepoxide (also known as Librium) is a widely prescribed minor tranquilizer. Four male Long-Evans hooded rats, maintained at 80% of their free-feeding weights of 325 to 375 g, were trained daily for 4 months to respond on a fixed-interval (FI 1) schedule to bar-press for a food pellet reward. At this time, a stable baseline pattern of performance was achieved, consisting of a positively accelerated rate of responding throughout each FI period until a food pellet was obtained. A dose-effect function was then established for chlordiazepoxide over the range 1 to 40 mg/kg. Chlordiazepoxide first increased the responding rate (approximately 2 to 3 times baseline at approximately 10

mg/kg), then caused the responding rate to decrease to approximately zero at 40 mg/kg. A similar dose-effect function was obtained with the same dose range of chlordiazepoxide, except that the rats were exposed to RFR during the 30-min period before the bar-pressing session. In this case, greater behavioral effects were found. The general shape of the dose-effect functions remained the same. However, the magnitude of the effect was increased, generally by a factor of two. Appropriate control runs were conducted to ensure stable and repeatable effects of each animal under the various dosing situations. Exposure to RFR alone produced no difference in the FI 1 responding rate.

FINAL CRITIQUE: The dose-response function for chlordiazepoxide was modified under pulsed RFR at an average power density of 1 mW/cm.2. The magnitude of the behavioral effect produced by the drug was strikingly potentiated by the RFR. The RFR alone had no effect on behavior. The mechanism of interaction of the RFR in this instance is not clear. The duty cycle of 0.001 yields a peak power density of 1 W/cm.2, which along with the pulse width of 2 microsec, indicates that the RFR hearing phenomenon (acoustic transients detectable by the animal) may have occurred. Likewise, although the incident average power density is low, regional SAR in the localized regions of the brain that are target areas for chlordiazepoxide's central actions may have been sufficiently high for a thermally potentiating effect. More research is clearly indicated on this effect, which occurs at what has previously been considered to be ineffectual levels of RFR exposure. A subsequent paper indicates that the potentiating effect is not obtained with two other drugs, chlorpromazine and diazepam (Thomas et al., 1980).

REFERENCES: Thomas, J. R., J. Schrot, and R. A. Banvard, "Behavioral Effects of Chlorpromazine and Diazepam Combined with Low-Level Microwaves," Neurobehav. Toxicol., Vol. 2, pp. 131-135 (1980)

(7) ENDOCRINOLOGICAL

List of Analyses

[2] Lancranjan, I., M. Maicanescu, E. Rafaila, I. Klepsch, and H. I. Popescu
GONADIC FUNCTION IN WORKMEN WITH LONG-TERM EXPOSURE TO MICROWAVES
Health Phys., Vol. 29, pp. 381-383 (1975)

[2] Lotz, W. G. and S. M. Michaelson
TEMPERATURE AND CORTICOSTERONE RELATIONSHIPS IN MICROWAVE-EXPOSED RATS
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IEEE Trans. Biomed. Eng., Vol. 24, No. 6, pp. 522-529 (1977a)

[2] Magin, R. L., S.-T. Lu, and S. M. Michaelson
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Radiat. Res., Vol. 62, pp. 225-241 (1975)
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[1] Sadchikova, M. N.
CLINICAL MANIFESTATIONS OF REACTIONS TO MICROWAVE IRRADIATION IN VARIOUS OCCUPATIONAL GROUPS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 261-267 (1974) (See "Epidemiologic" for analysis.)

Analyses under

(7) ENDOCRINOLOGICAL

Lancranjan, I., M. Maicanescu, E. Rafaila, I. Klepsch, and H. I. Popescu
GONADIC FUNCTION IN WORKMEN WITH LONG-TERM EXPOSURE TO MICROWAVES
Health Phys., Vol. 29, pp. 381-383 (1975)

Study type: (7) Endocrinological, (1) Epidemiological, (6) Behavioral; IN VIVO; HUMAN

Effect type: RFR-induced alterations of libido, semen characteristics, and 17-ketosteroid and gonadotropin contents in urine

Frequency/wavelength: 3.6-10 GHz/3-12 cm

Modulation: Not stated

Power Densities: Tens to hundreds of microwatts/cm.2

SAR: Not measured

Exposure conditions: Various occupational situations, some with shielding. Exposure durations ranged from 1 to 17 yrs (a mean of 8 yrs)

AUTHOR ABSTRACT: A study was carried out in 31 young men (mean 33 yr) with long-term exposure (mean 8 yr) to microwaves. The investigation included a detailed andrologic questionary (sic) and semen analyses as well as determinations of total neutral 17-ketosteroids (17 ks) and total gonadotropin (t.g.) eliminations in 24-hr urine. The investigation showed a high frequency of libido decrease and sexual dynamic disturbances in the framework of the asthenic syndrome (70% of subjects) as well as various alterations of spermatogenesis in 74% of the subjects. The alterations consisted of asthenospermia, hypospermia and/or teratospermia. The function of the testes leydigian cells, indirectly indicated by 17 ks determination, was normal. The normal or increased values of t.g. eliminations ruled out the possibility of the contribution of a hypothalamo-pituitary imbalance in the induction of germinal alterations shown in exposed men.

OTHER INFORMATION: The mean age of the 31 exposed subjects was 33 yrs; 28 of them were less than 40 yrs old. Potential subjects suffering from endocrinologic diseases, venereal infections, varicocel, hydrocel, trauma of the testes, and other conditions affecting spermatogenesis were eliminated by questionnaire. Semen volume was measured and counts of total, mobile, and abnormal sperm per ml were made for each subject

after at least 3 days of sexual inactivity. In addition, the total neutral 17-ketosteroids (17 ks) and total gonadotropins (t.g.) in 24-hr urine were determined for 19 of the subjects. Controls consisted of 30 unexposed men (mean age 34 yrs) for the semen analyses, 8 for the 17 ks analyses, and 10 for the t.g. analyses. The authors state that 23 (74%) of the exposed subjects exhibited alterations of spermatogenesis, but provide no details except mean values for the entire group relative to those for the controls. The mean total sperm count for the subjects was 50 million/ml, with a standard deviation of 24. The corresponding figures for the controls were 60 plus or minus 34 million/ml. The authors state that based on the t-test, the difference is significant (p less than 2%), but a calculation shows that t is 1.33, so that p is greater than 10% for the 2-tailed test or greater than 5% for the 1-tailed test. The mean numbers of motile and normal sperm for the exposed group were 36 plus or minus 10 and 70 plus or minus 6, respectively, and the corresponding values for the control group were 54 plus or minus 19 and 82 plus or minus 7 (millions/ml). Both differences were statistically significant (p less than 0.001), as stated by the authors. There was no significant difference between the mean 17 ks elimination for the 19 exposed subjects and the 8 controls. Also, 14 subjects had normal values of t.g. elimination, 4 had higher values, and one had a value below the lower limit of the normal range; this man (age 33 yrs) had been exposed to RFR for 14 yrs and also exhibited azoospermia. The authors interpreted the normal and higher values of t.g. as ruling out the possibility that spermatogenesis alterations could be secondary to a central diencephalo-pituitary disorder inducing a decrease in gonadotropic stimulation, but rather as due to the direct effect of RFR on the germinal epithelium of the gonads. The authors also stated that 80% of the exposed subjects suffered from asthenic syndrome, which included complaints of decreases of libido and of erection, ejaculation, and/or other orgasm disturbances, and indicated that reinvestigation after 3 months of interruption of RFR exposure showed improved spermatogenesis in two-thirds of the subjects, but gave no data.

FINAL CRITIQUE: As with other studies of the effects of occupational exposure to RFR, accurate information is lacking on intensities and durations of exposure. The authors mentioned that the intensity "varied according to the working process, the existence of some shielding systems, and the power installations as well," but studied only 31 subjects without distinction among the various exposure situations. Moreover, it is difficult to determine the influence of physiological and emotional factors not related to RFR exposure in the occupational environment or the personal lives of the subjects,

especially with so few. These factors could adversely affect spermatogenesis and give rise to the subjective complaints mentioned. In addition, removal from the occupational environment for 3 months could indeed produce improvement. Thus, the significance of these findings is conjectural.

Lotz, W. G. and S. M. Michaelson
TEMPERATURE AND CORTICOSTERONE RELATIONSHIPS IN MICROWAVE-EXPOSED RATS
J. Appl. Physiol.: Respirat. Environ. Exercise Physiol., Vol. 44, No. 3, pp. 438-445 (1978)

Study type: (7) Endocrinological; IN VIVO; RAT

Effect type: RFR-induced changes in plasma corticosterone levels and colonic temperature

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 0 to 60 mW/cm.2

SAR: 0.16 W/kg per mW/cm.2

Exposure conditions: In groups of four for 30, 60, or 120 min at 0, 13, 20, 30, or 40 mW/cm.2 or for 30 or 60 min at 50 or 60 mW/cm.2 in an anechoic chamber held at 24 deg C and 40 to 60% relative humidity. Two controls for each exposure were sham-exposed under similar conditions

AUTHOR ABSTRACT: Plasma corticosterone and colonic temperature were measured in unanesthetized male rats exposed to 2,450-MHz continuous wave (cw) radiation to characterize the response of the pituitary-adrenal axis to microwave exposure. The rats were exposed in the far field of a horn antenna for 30 or 60 min at power densities of 0, 13, 20, 30, 40, 50, or 60 mW/cm.2, or for 120 min at 0, 13, 20, 30, or 40mW/cm.2. The average energy absorption rate of the rats was 0.16 W/kg absorbed per mW/cm.2 incident. Plasma from individual rats decapitated immediately after exposure was collected for analysis. Colonic temperature was significantly elevated after exposures to power densities of 13 mW/cm.2 or greater, with progressively larger increases after high intensity exposures. Plasma corticosterone was significantly elevated above control levels only after exposures at 50 or 60 mW/cm.2 for 30- or 60-min exposures, and at 20, 30, and 40 mW/cm.2 for 120-min exposures. The relationship between the increased levels of circulating corticosterone and colonic temperature suggested that the increases in corticosterone levels may reflect a level of physiological response to the body temperature elevations caused by microwave exposure.

OTHER INFORMATION: SARs were determined calorimetrically in water phantoms, and found to be 0.16 W/kg per mW/cm.2. The

mean resting metabolic rate for 4 rats was calculated from measurements of their oxygen consumption and found to be 7.6 W/kg. All rats were "gentled" for 2 weeks prior to exposure by weighing and handling them at least 4 times per week. Each rat was conditioned for exposure by taking its colonic temperature and placing the rat in an exposure cage for 3 to 5 hrs on 3 of the last 4 days before use. The activity of the adrenal axis during the 3-hr equilibration period prior to exposure was determined by placing groups of rats in the exposure chamber for 30, 60, 90, 120, 150, or 180 min, measuring their colonic temperature before and after this interval, and assaying their blood for corticosterone. The results indicated a rapid rise of both colonic temperature and corticosterone during the first half hour to an approximate plateau, followed by a return to baseline values by the end of 3 hrs, demonstrating the need for such an equilibration period prior to exposure. Groups of 4 rats each were exposed to RFR for 30 or 60 min at 13, 20, 30, 40, 50, or 60 mW/cm.2 or for 120 min at 13, 20, 30, or 40 mW/cm.2. Two sham-exposed rats per group served as controls. Colonic temperatures and corticosterone levels were determined. Plots of colonic temperature versus exposure duration at the various power densities showed a small but statistically significant temperature rise after 30-min exposure to 13 mW/cm.2; exposures for the same duration to the higher power densities produced temperature increases approximately proportional to the power density. Up to 40 mW/cm.2, 60-min exposures produced temperature increases not significantly greater than those for 30 min, but the temperature rises for 60-min exposures at 50 or 60 mW/cm.2 were significantly larger than those for 30 min. Plasma corticosterone levels were more widely scattered. At each power density, increases in mean level with duration were discernible, but the results were not significantly different from baseline values for durations of up to 120 min at 13 mW/cm.2, up to 60 min at 20 mW/cm.2, and 30 min at 30 mW/cm.2, indicative of a threshold pattern of response. All other increases were significant. However, the four mean values obtained from 30- and 60-min exposures at 30 and 40 mW/cm.2, though larger than baseline, were not significantly different from one another. The increases of corticosterone level were highly correlated with the rises in colonic temperature, indicating that the former may be a concomitant of the latter. Threshold values for adrenal-axis stimulation were estimated to be about 30-50 mW/cm.2 for 60-min exposure, and 15-20 mW/cm.2 for 120-min exposure. Using the SARs measured in water phantoms yields thresholds of 4.8-8.0 W/kg for 60-min exposure, and 2.4-3.2 W/kg for 120-min exposure. The latter range is somewhat less than half the resting metabolic rate.

FINAL CRITIQUE: A major point demonstrated in this investigation is the necessity for equilibrating rats for at least 3 hrs prior to exposure, to minimize the non-RFR stresses imposed on them in the experimental situation. The authors also took pains to perform the RFR- and sham-exposures during the same period each day, and to exsanguinate the animals and preserve the blood as quickly as possible after exposure for corticosterone assay. The relatively large scatter in corticosterone levels in each group was therefore probably due to individual differences among the animals rather than to methodology-related variations. Despite the scatter, the mean corticosterone levels were highly correlated with colonic temperatures. Also, the results do indicate the existence of RFR thresholds. However, the estimated threshold value for 120-min exposure is lower than that for 60 min, and the authors are cautious about extrapolating their results to exposures of longer duration. (Regarding the 2-hr threshold, a minor inconsistency should be noted: on p. 442, the authors cite the range as 15-20 mW/cm.2, whereas on p. 443, the range cited is 20-30 mW/cm.2, but this discrepancy does not affect the conclusions.)

Lu, S.-T., N. Lebda, S. M. Michaelson, S. Pettit, and D. Rivera
THERMAL AND ENDOCRINOLOGICAL EFFECTS OF PROTRACTED IRRADIATION OF RATS BY 2450-MHZ MICROWAVES
Radio Sci., Vol. 12, No. 6S, pp. 147-156 (1977)

Study type: (7) Endocrinological,
(9) Biochemical/physiological; IN VIVO; RAT

Effect type: RFR-induced changes in whole-body mass; normalized mass of the pituitary, adrenal, and thyroid glands; and changes in serum levels of corticosteroid, thyroxine, growth hormone.

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 0, 1, 5, 10, or 20 mW/cm.2

SAR: 5 W/kg at 20 mW/cm.2

Exposure conditions: 4 rats in separated individual styrofoam cages were exposed, after 3 hrs equilibration, to far-field RFR at one of the stated power densities. For controls, 1 or 2 rats were sham-exposed for each duration. Chamber was maintained at 24 deg C. All exposures and sham exposures were performed on Thursdays at 1130.

AUTHOR ABSTRACT: Eighty-six male Long-Evans rats, 63-64 days of age, were subjected to 2450-MHz CW microwave irradiation after a two-week "gentling" procedure and three daily sessions of sham exposure. Exposures occurred without anesthesia at power densities of 0, 1, 5, 10, or 20 mW/cm.2 for 1, 2, 4, or 8 hours. Rectal temperatures of exposed rats were found to be higher than those of sham-irradiated rats at power densities of 1 mW/cm.2 for four hours, 5 mW/cm.2 for one and two hours, 10 mW/cm.2 for two and four hours, and 20 mW/cm.2 for all of the durations of exposure. Circadian rhythmicity of rectal temperature was noted in sham-irradiated rats. Except for rats exposed at 20 mW/cm.2 for four and eight hours, none of the averaged rectal temperatures of exposed groups reliably exceeded those of sham-irradiated rats after eight hours of exposure. We concluded that the effect of exposure to microwaves at power densities below 10 mW/cm.2 accelerated the appearance of the peak rectal temperature to an earlier time of day. Serum corticosteroid (CS) levels were significantly lower in rats exposed at 20 mW/cm.2 for eight hours than in animals sham exposed for the same duration. A significant correlation between rectal temperature and CS level was observed in the

sham-irradiated rats. Certain combinations of duration of exposure and power density of incident energy could dissociate this relationship, i. e., temperature increased sometimes without corresponding elevation of CS level. Serum thyroxine levels were depressed in rats exposed to radiation at 20 mW/cm.2 for four or for eight hours. A stimulatory effect of microwaves on thyroid function was noted in rats exposed at 1 mW for four hours, but the effect was transitory. Levels of growth hormone did not change.

OTHER INFORMATION: Several days after arrival, the rats were subjected to gentling procedures for 2 weeks, including removal from the cage, taking of rectal temperature, and measurement of body mass. During the following week (at age 61-62 days) they were sham-exposed daily on 3 successive days (Mon-Wed). Each session included measurement of body mass and rectal temperature, transfer to exposure cage for 3 hrs, subsequent measurement of body mass and rectal temperature, and transfer back to home cage. Rats were then exposed or sham-exposed on the following day (Thurs), after the 3-hr equilibration period. After exposure, each rat was decapitated, blood was collected, and body mass and rectal temperature were measured. Also, the pituitary, adrenal, and thyroid glands were removed, fixed, and weighed. Serum levels of corticosteroid (CS), thyroxine (T-4), and growth hormone (GH) were assayed. Rectal temperatures were found to vary widely during the 2-week gentling period and during each day, the latter variations including those related to the circadian period. Mean rectal temperatures for the sham-exposed rats increased with exposure duration (1, 2, 4, and 8 hrs), ascribed to circadian rhythmicity. For 1-hr RFR exposures, significant increases in rectal temperature relative to the mean for the sham-exposed group were noted for the groups exposed at 5 and 20 mW/cm.2, but not at 1 and 10 mW/cm.2. For those exposed for 2 hrs, significant temperature increases were obtained at 5, 10, and 20 mW/cm.2; for 4-hr exposures, the increases were significant at 1, 10, and 20 mW/cm.2, and for 8-hr exposures they were significant only at 20 mW/cm.2. For the sham-exposed animals, the CS level increased with exposure duration and was correlated with the rectal temperature increase. Similarly, the CS level increased, with few exceptions, with RFR-exposure duration at all power densities, but there was no significant correlation with rectal temperature. The only statistically significant changes in serum thyroxine level were an increase for 4-hr exposure at 1 mW/cm.2 and decreases for 4-hr and 8-hr exposures at 20 mW/cm.2. No significant changes in GH level were noted. There were no significant alterations of body mass due to RFR exposure or of pituitary mass (normalized to body mass). Several statistically significant alterations of thyroid and

adrenal mass were noted. There were no significant alterations of body mass due to RFR exposure or of pituitary mass (normalized to body mass). Several statistically significant alterations of thyroid and adrenal mass were noted among the RFR- and sham-exposed groups, but with no obvious pattern related to power density, exposure duration, or circadian rhythmicity.

FINAL CRITIQUE: In view of the large scatters of values of each endpoint among the animals sham-exposed for various durations (presumably due to unknown differences in residual stress reactions after gentling as well as the circadian variations discussed by the authors), it is difficult to discern any clear-cut effects ascribable to RFR exposure per se. The authors in recognizing such difficulties state that "In spite of these constraints, we were able to show that exposure of rats to 2450-MHz CW microwaves at 20 mW/cm.2 for eight hours depressed serum CS levels." Indeed their results show a statistically significant lower mean CS value for the 4 rats thus exposed than for the 6 rats that were sham-exposed for the same duration. Also, comparisons between groups exposed to RFR at other power densities and groups sham-exposed for the same durations showed both increases and decreases of CS level, but none of these changes were statistically significant. However, large changes (primarily increases) in CS level with exposure duration at constant power density were evident for most groups, including those that were sham-exposed. Therefore, possible RFR-induced changes, if any, would probably be masked by the non-RFR-induced changes, and the significant result cited above was probably a statistical anomaly. In general, the number of rats used for each exposure regimen was too small to lend much biological credence to any of the statistically significant differences between RFR- and sham-exposed rats. It should be noted that the authors used the Mann-Whitney U-test on the CS values and the Student t-test on the rectal temperatures without indicating their justification for doing so. This point is mentioned because had they used the t-test on the 20-mW/cm.2, 8-hr CS data relative to the corresponding controls, they would have found the CS depression to be non-significant.

Magin, R. L., S.-T. Lu, and S. M. Michaelson
MICROWAVE HEATING EFFECT ON THE DOG THYROID GLAND
IEEE Trans. Biomed. Eng., Vol. 24, No. 6, pp. 522-529 (1977a)

Study type: (7) Endocrinological; IN VIVO; DOG

Effect type: Increases in release rates of thyroxine (T4) and triiodothyronine (T3) due to RFR heating of the thyroid only

Frequency/wavelength: 2.45 GHz

Modulation: 120 Hz

Power Densities: 72, 162, and 236 mW/cm.2

SAR: 58, 131, and 190 W/kg

Exposure conditions: Irradiation of one surgically exposed thyroid with a diathermy unit and special applicator for 2 hrs after 1-hr equilibration to establish baseline hormone release rates; the other thyroid, also surgically exposed, served as control.

AUTHOR ABSTRACT: Dog thyroid glands were exposed IN VIVO to 2450 MHz, CW microwave fields for 2 h using a dielectrically loaded waveguide applicator. Specific absorption rates of 58, 131, and 190 W/kg in the center of the thyroid gland resulted in temperatures of 38-39 C, 40-42 C, and 44-46 C, respectively. An increase in the heated gland's thyroid hormone, thyroxine (T4) and triiodothyronine (T3), release rate was observed. This result demonstrates that the dog thyroid gland can be directly stimulated by microwave heating.

OTHER INFORMATION: See also Magin et al. (1977b), which describes the same investigation but includes more detailed discussion of other aspects. Both thyroid glands were exposed so that the caudal thyroid veins draining each gland could be isolated. The internal jugular veins were ligated above and cannulated below the junction with the caudal thyroid veins. Veins of non-thyroidal origin draining into the internal jugular were also ligated. In addition, a vein that connects the two thyroid glands across the trachea was obliterated. The first hour after cannulation was used as an equilibration period to establish the baseline hormone release rate for each gland. During the subsequent 2 hrs, one gland was locally heated with RFR while the other was used as its control. Blood was collected continuously for all 3 hrs in 20-min sampling periods. A femoral artery blood sample was collected at the middle of each sampling period and the mean arterial blood

pressure and heart rate were measured after sample collection. Saline and heparin were infused to maintain blood volume and prevent clotting. Concentrations of thyroxine (T4) and triiodothyronine (T3) were measured and the hormonal release rate of each gland was calculated from the product of the difference in hormonal concentration between the cannulated-vein and femoral-artery samples, the thyroid blood-flow rate, and the hematocrit (ratio of plasma to whole blood). The results were expressed as the ratio (in percent) of hormone released during exposure to that released during equilibration as a function of time after cannulation. For the sham-exposed experiments, the mean T4 release was about 100%, with a standard error of about 50%. Thyroid temperature rises at several values of net forward power applied for 10 s were used to determine the SAR per watt of forward power. Three groups of RFR exposures were then performed, in which the forward power was varied during each exposure to maintain thyroid temperature in the ranges 38-39, 40-42, or 44-46 deg C. (The baseline temperature of thyroids open to ambient conditions was about 36 deg C.) The mean values of SAR were those cited in the abstract. Rectal and tympanic-membrane temperatures (the latter a reliable estimate of the hypothalamic temperature) were also monitored. The results for the 38-39 deg group showed a step increase in T4 release to about 200%, indicative of a general increase in glandular metabolism due to the heat. The curve paralleled the step in gland temperature beginning at the onset of RFR exposure. For the other two groups, the T4 release rate peaked after about 50 min of RFR exposure (at about 300% for the intermediate group and 600% for the upper group) and diminished thereafter. This behavior was ascribed to either thermal damage or depletion of readily releasable hormone. However, histological examination of the thyroid glands after RFR exposure showed no apparent structural changes or degeneration.

FINAL CRITIQUE: The results of this investigation show that local heating of the thyroid by RFR increases the blood-flow rate and hormone release rate in the absence of any RFR stimulation of the hypothalamic-hypophysial axis. These findings are significant because the human neck is a region of high local SAR relative to the whole-body SAR for specific frequency ranges and exposure conditions (Gandhi, 1975; Guy et al., 1976), so that such heating could occur for even relatively moderate incident power densities.

REFERENCES: Gandhi, O. P., CONDITIONS OF STRONGEST ELECTROMAGNETIC POWER DEPOSITION IN MAN AND ANIMALS, IEEE Trans. Microwave Theory Tech., Vol. 23, No. 12, pp. 1021-1029 (1975)

Guy, A. W., M. D. Webb, and C. C. Sorensen, DETERMINATION OF POWER ABSORPTION IN MAN EXPOSED TO HIGH FREQUENCY ELECTROMAGNETIC FIELDS BY THERMOGRAPHIC MEASUREMENTS ON SCALE MODELS, IEEE Trans. Biomed. Eng., Vol. 23, No. 5, pp. 361-371 (1976)

Magin, R. L., S.-T. Lu, and S. M. Michaelson, STIMULATION OF DOG THYROID BY LOCAL APPLICATION OF HIGH INTENSITY MICROWAVES, Am. J. Physiol., Vol. 233, No. 5, pp. E363-E368 (1977b)

Magin, R. L., S.-T. Lu, and S. M. Michaelson
STIMULATION OF DOG THYROID BY LOCAL APPLICATION OF HIGH INTENSITY MICROWAVES
Am. J. Physiol., Vol. 233, No. 5, pp. E363-E368 (1977b)

Study type: (7) Endocrinological,
(9) Biochemical/physiological; IN VIVO; DOG

Effect type: Increases in thyroid blood flow, thyroxine (T4) concentration and release rate, and thyroid metabolic rate due to RFR heating of the thyroid only

Frequency/wavelength: 2.45 GHz

Modulation: 120 Hz

Power Densities: 72, 162, and 236 mW/cm.2

SAR: 58, 131, and 190 W/kg

Exposure conditions: Irradiation of one surgically exposed thyroid with a diathermy unit and special applicator for 2 hrs after 1-hr equilibration to establish baseline hormone release rates; the other thyroid, also surgically exposed, served as control

AUTHOR ABSTRACT: Microwave radiation (2.45 GHz, CW) was applied locally to one of a dog's paired thyroid glands. The dog was anesthetized with sodium pentobarbital so that both glands could be surgically exposed and their caudal veins cannulated. The function of the thyroid glands was assessed by the measurement of total plasma thyroxine (T4) in collected blood samples using a radioimmunoassay. The cannulated glands were allowed a 1-h equilibration period followed by a 2-h exposure period during which the temperature of the heated gland was maintained at approximately 39, 41, or 45 deg C. The thyroxine release rate (ng T4/min) was determined from the difference between the T4 concentration in the thyroid veins and femoral artery, the thyroid vein blood flow, and the hematocrit. The thyroxine release rate was increased during the exposure period to 150, 350, and 1000% of the equilibration period means for the three temperatures studied. The blood flow was increased to 140 and 170% of the equilibration period means in the glands heated to 41 and 45 deg C. The contralateral control glands and sham-exposed glands maintained a constant thyroxine release rate. The results demonstrate that local heating of the thyroid gland can stimulate both thyroid gland blood flow and the release of thyroxine.

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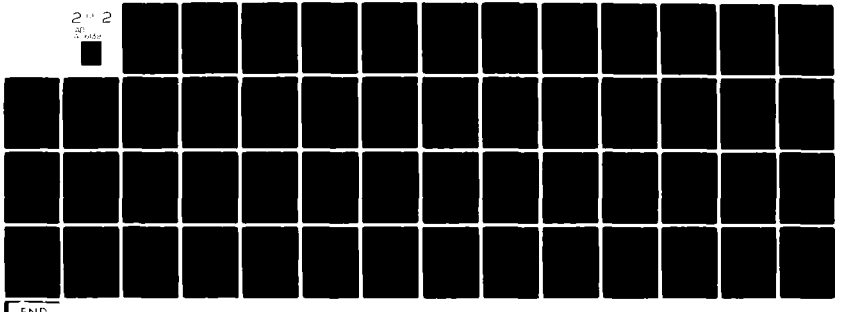

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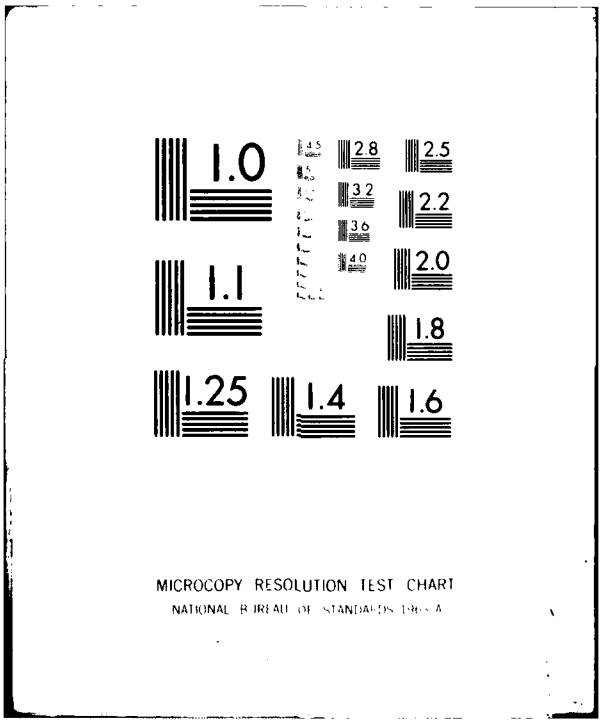
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MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS 1963-A

OTHER INFORMATION: See also Magin et al. (1977a), which describes the same investigation but includes more detailed discussion of other aspects. Both thyroid glands were exposed so that the caudal thyroid veins draining each gland could be isolated. The internal jugular veins were ligated above and cannulated below the junction with the caudal thyroid veins. Veins of non-thyroidal origin draining into the internal jugular were also ligated. In addition, a vein that connects the two thyroid glands across the trachea was obliterated. The first hour after cannulation was used as an equilibration period to establish the baseline hormone release rate for each gland. During the subsequent 2 hrs, one gland was locally heated with RFR while the other was used as its control. Blood was collected continuously for all 3 hrs in 20-min sampling periods. A femoral artery blood sample was collected at the middle of each sampling period and the mean arterial blood pressure and heart rate were measured after sample collection. Saline and heparin were infused to maintain blood volume and prevent clotting. Concentrations of thyroxine (T4) were measured, and the difference between the cannulated-vein and femoral-artery concentration was determined for each sample. The results for each gland over the equilibration period were averaged, and the results for the subsequent samples were expressed as percentages of this average. In addition, the thyroxine release rate was calculated from the product of the concentration difference, the thyroid blood-flow rate, and the hematocrit (ratio of plasma to whole blood) as a function of time after cannulation. Three groups of RFR exposures were performed, in which the power was varied during each exposure to maintain substantially constant thyroid temperature. The mean values for the three groups were 39.4, 41.4, and 44.8 deg C, representing increases of 2.8, 4.6, and 8.7 deg C above the respective mean equilibration mean values. For the sham-exposed glands and their contralateral controls, the mean temperature was about 35.6 deg C. The tympanic-membrane and rectal temperatures remained substantially constant for the three RFR-exposed and the sham-exposed groups. The T4 concentration differences were found to increase during the 3-hr experiments not only for the thyroid groups exposed to RFR but also for the sham-exposed and control groups. However, the blood-flow measurements for the latter two groups showed decreases to about half the equilibration mean values by the end of the 3-hr period. Also, the hematocrit decreased slightly because of saline infusion. Thus, the calculated T4 release rates were substantially constant during the period for these two groups. Similar calculations for the 39.4-deg group yielded a steplike increase in the T4 release rate to about 200% of the equilibrium value at the onset of RFR exposure. For the 41.4 and 44.8-deg groups, the increases were to 350%

and 1000%, respectively, by about 1-hr into the exposure period, but diminished thereafter. These increases were due to heat-induced increases in metabolic rate as well as bloodflow; the subsequent declines in the two higher-temperature groups were ascribed to either depletion of the readily releasable supply of T4 or thermal damage.

FINAL CRITIQUE: The results of this investigation show that local heating of the thyroid by RFR increases the blood-flow rate and hormone release rate in the absence of any RFR stimulation of the hypothalamic-hypophysial axis. These findings are significant because the human neck is a region of high local SAR relative to the whole-body SAR for specific frequency ranges and exposure conditions (Gandhi, 1975; Guy et al., 1976), so that such heating could occur for even relatively moderate incident power densities.

REFERENCES: Gandhi, O. P., CONDITIONS OF STRONGEST ELECTROMAGNETIC POWER DEPOSITION IN MAN AND ANIMALS, IEEE Trans. Microwave Theory Tech., Vol. 23, No. 12, pp. 1021-1029 (1975)

Guy, A. W., M. D. Webb, and C. C. Sorensen, DETERMINATION OF POWER ABSORPTION IN MAN EXPOSED TO HIGH FREQUENCY ELECTROMAGNETIC FIELDS BY THERMOGRAPHIC MEASUREMENTS ON SCALE MODELS, IEEE Trans. Biomed. Eng., Vol. 23, No. 5, pp. 361-371 (1976)

Magin, R. L., S.-T. Lu, and S. M. Michaelson, MICROWAVE HEATING EFFECT ON THE DOG THYROID GLAND, IEEE Trans. Biomed. Eng., Vol. 24, No. 6, pp. 522-529 (1977a)

(8) IMMUNOLOGICAL

List of Analyses

[2] Czerski, P.
MICROWAVE EFFECTS ON THE BLOOD-FORMING SYSTEM WITH PARTICULAR REFERENCE TO THE LYMPHOCYTE
Ann. N.Y. Acad. Sci., Vol. 247, pp. 232-242 (1975)

[2] Hamrick, P. E., D. I. McRee, P. Thaxton, and C. R. Parkhurst
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[2] Huang, A. T., M. E. Engle, J. A. Elder, J. B. Kinn and T. R. Ward
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FOREIGN SERVICE HEALTH STATUS STUDY: EVALUATION OF HEALTH STATUS OF FOREIGN SERVICE AND OTHER EMPLOYEES FROM SELECTED EASTERN EUROPEAN POSTS
Final Report, July 31, 1978, Contract No. 6025-619073, Dept. of Epidemiology, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, MD (1978) (See "Epidemiologic" for analysis.)

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0048-6604/79/1112-S026, Radio Sci., Vol. 14, No. 6S, pp. 173-179 (1979) (See "Biochemical/Physiological" for analysis.)

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Radiat. Res., Vol. 71, No. 2, pp. 355-366 (1977) (See "Teratogenic and Developmental Abnormalities" for analysis.)

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[2]Stodolnik-Baranska, W.
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In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 189-195 (1974)

[2]Wiktor-Jedrzejczak, W., A. Ahmed, P. Czerski, W. M. Leach, and K. W. Sell
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[2]Wiktor-Jedrzejczak, W., A. Ahmed, P. Czerski, W. M. Leach, and K. W. Sell
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Radio Sci., Vol. 12, No. 6S, pp. 209-219 (1977)

Analyses under

(8) IMMUNOLOGICAL

Czerski, P.
MICROWAVE EFFECTS ON THE BLOOD-FORMING SYSTEM WITH PARTICULAR REFERENCE TO THE LYMPHOCYTE
Ann. N.Y. Acad. Sci., Vol. 247, pp. 232-242 (1975)

Study type: (8) Immunological; IN-VIVO; MOUSE and RABBIT; IN-VITRO HUMAN lymphocyte cultures

Effect type: RFR-induced lymphoblastoid transformations in immunocompetent animals

Frequency/wavelength: 2950 MHz

Modulation: 1 microsecond pulses, 1200 pps

Power Densities: 0.5 mW/cm.2 for mice; 5 mW/cm.2 for rabbits

SAR: Not measured

Exposure conditions: 2 hrs/day, 6 days/week in the far field, IN-VIVO for 6 or 12 weeks for mice and 6 months for rabbits; IN-VITRO exposures of cultures not specified

REVIEWER SUMMARY: One hundred mice were exposed to pulsed 2950-MHz RFR at 0.5 mW/cm.2 average power density for 6 weeks, and another 100 for 12 weeks (2 hr/day, 6 days/week). After exposure the mice were immunized by injection of 200 million sheep red blood cells (SRBC). For controls, 100 unexposed mice were immunized and two other groups of 100 each were exposed for 6 or 12 weeks but not immunized. Five mice from each group were then euthanized on days 4, 6, 8, 12, and 20. Sera were collected, lymph nodes were excised, suspensions of cells therefrom were prepared, and the percentages of lymphoblasts and plasmocytes in the latter were determined. The number of antibody-forming cells was determined as a measure of the immune response. Also, serum hemagglutinins were estimated. For the 3 immunized groups, the percentage of blast cells rose from an initial value (of about 6%), reached maxima on day 6 after injection of SRBC, and diminished to approximately baseline values by day 20. The smallest maximum, about 27%, was for the unexposed group; the next, about 32%, was for the group exposed for 12 weeks; and the highest, about 50%, was for those exposed for 6 weeks. Smaller rises to maxima on day 6 were obtained for the two nonimmunized groups, with the 6-week group again yielding a higher maximum than the 12-week group. Qualitatively similar patterns were obtained for the percentage of plasmocytes and the number of antibody-producing cells. In another series, 12 rabbits were exposed to the same RFR at 5 mW/cm.2 for 6 months. Each month, peripheral blood

was obtained, lymphocyte cultures were prepared and incubated for 7 days at 37 deg C, and the percentage of blast cells was counted. The results showed an increase from about 3% initially to 9 and 10% respectively for months 1 and 2 of exposure, after which the percentage returned to baseline. A smaller increase, to about 6%, was also seen for month 7 (1 month after cessation of exposure) and a return to baseline for months 8 and 9. Preliminary work was also done with human lymphocyte cultures exposed IN-VITRO to RFR (power densities and durations not specified) toward reproducing the results of Stodolnik-Baranska (1974). Unexposed samples or samples incubated for 1 hr with phytohemagglutinin (PHA) served as controls. After the cultures were exposed to RFR and incubated for 72 hrs, the percentage of blast cells was determined. Also, after incubation for 24 hrs, cultures were used to test for macrophage migration inhibition in mouse spleen fragments. Although a few positive results were obtained, the data were poorly reproducible.

FINAL CRITIQUE: Most of the results were presented graphically; no error bars were given and statistical treatment of the data was not discussed, rendering it difficult to ascertain which of the differences described were statistically significant. Also, it is not clear from the text whether the unexposed animals were sham-irradiated. Perhaps the most interesting observation with the mice was that the group exposed for 6 weeks and immunized had higher maximum percentages of blast cells, plasmocytes, and antibody-producing cells than the group exposed for 12 weeks and immunized. The author surmised that this result was an indication of adaptation to the RFR, a hypothesis that is qualitatively supported by the results with the rabbits. However, it should be noted that the titer of serum hemagglutinins for both groups of exposed, immunized mice rose essentially monotonically at the same rate through day 20. Also interesting is that the maximum percentage of blast cells for the immunized unexposed group was almost as high as for the group exposed for 12 weeks and immunized, that the maxima for all three immunized groups occurred on day 6 after injection of SBRC, and that the maximum for the 6-week group not immunized also occurred on day 6 even though no SBRC was administered. In general, the results of this investigation indicate that IN-VIVO exposure to RFR at the frequency and power densities used does alter the immune system, but the significance of such findings with respect to possible effects of RFR on the human immune system is difficult to assess. The difficulties in reproducing the results of Stodolnik-Baranska (1974) from IN-VITRO exposures of human lymphocyte cultures to RFR render such results even more problematical.

REFERENCES: Stodolnik-Baranska, W., THE EFFECTS OF MICROWAVES ON HUMAN LYMPHOCYTE CULTURES, in P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 189-195 (1974).

Hamrick, P. E., D. I. McRee, P. Thaxton, and C. R. Parkhurst
HUMORAL IMMUNITY OF JAPANESE QUAIL SUBJECTED TO MICROWAVE RADIATION DURING EMBRYOGENY
Health Phys., Vol. 33, pp. 23-33 (1977)

Study type: (8) Immunological, (3) Teratogenic and developmental abnormalities, (9) Biochemical/physiological; IN VIVO; JAPANESE QUAIL

Effect type: Reduced immunocompetence of birds induced by RFR exposure of eggs

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 5 mW/cm.2

SAR: 4.03 W/kg

Exposure conditions: 6X5 arrays of eggs in far field in chamber at 35.5 deg C and 60% relative humidity. Each array was exposed 24 hrs/day for the first 12 days of development. Arrays were automatically turned 90 deg every 2 hrs. Control arrays were sham-exposed but otherwise treated similarly.

AUTHOR ABSTRACT: Fertile Japanese quail eggs were exposed to continuous wave microwave radiation at an intensity of 5 mW/cm.2 (50 W/m.2) and a frequency of 2450 MHz. The absorbed power density was determined to be 4.03 W/kg. The eggs were exposed throughout the first 12 days of the normal incubation period of 17.5 days. Non-exposed control eggs were incubated in a chamber identical to the exposure chamber. After hatching, exposed and control quail were reared in the conventional laboratory manner. Weekly body weight measurements were made to compare the growth patterns of exposed and control quail. The weights of the exposed males at the ages of 4 and 5 weeks were 12 and 7%, respectively, less than the control males. These differences approached statistical significance (P less than or equal to 0.05). At 5 weeks of age the quail were challenged with sheep red blood cells (SRBC) and the levels of the anti-SRBC antibodies were determined. The levels of specific anti-SRBC antibodies, determined 4 days after antigen challenges, were of the same magnitude for both the exposed and control quail. Following this assessment of humoral immunity, the quail were sacrificed and the bursa of Fabricius and spleen were removed and a comparison was made of exposed and control birds. The weights

of the bursa of Fabricius and spleen were not altered significantly by the microwave exposure.

OTHER INFORMATION: The authors stated that each array was exposed with the major axes of the eggs parallel to the electric field (vertical). However, they also stated that each array was automatically turned 90 deg every 2 hrs, implying that the eggs were exposed with their major axes parallel to the magnetic field (horizontal) for half the time. Temperature distributions along the central major axis of one of the eggs in an array (with major axes vertical) and along the minor axis parallel to the propagation direction (front-to-rear) were measured with a thermistor showed to be nonperturbing. The results showed approximately constant temperature along the vertical axis, but the center was found to be 0.4 deg C hotter than the front and 0.6 deg C hotter than the rear. (Analogous measurements for horizontal exposure were not presented.) Temperatures of the eggs in an array exposed to RFR with the chamber at 35.5 deg C ranged from 37.5 to 38.0 deg C. Four arrays of 30 eggs each were exposed for the first 12 days (24 hrs/day) of development and transferred to a normal hatching incubator for the remainder of the 16- to 17-day incubation period. Control arrays were sham-irradiated and incubated for the same periods. After hatching, the chicks were maintained at a brooding temperature of 35 deg C for the first week and 30 deg C for the next two weeks, and thereafter at an ambient temperature of about 25 deg C. The quail were weighed at hatching and weekly afterward. Mortality was recorded daily. After five weeks, each quail was immunized with sheep red blood cells (SRBC). The quail were bled immediately before, and 4 days after, immunization, and serum anti-SRBC hemagglutinins were assayed. After the fourth-day bleeding, the quail were euthanized and the bursae of Fabricius and the spleens were weighed. The numbers of quail involved were 29 males and 48 females from eggs exposed to RFR, and 21 males and 19 females from sham-exposed eggs. Statistical treatment of the data showed no significant differences in mortality or in mean body weights between exposed and control groups of the same sex, but the females of both groups were heavier than their male counterparts. There were no significant differences in the weights of the bursa of Fabricius or the spleen irrespective of sex or in the mean anti-SRBC antibody levels prior to and 4 days after immunization.

FINAL CRITIQUE: These results provide no evidence that exposure of quail eggs to 2.45 GHz RFR at 5 mW/cm.2 increases the mortality of hatched birds or significantly alters their mean weights or their immunocompetence relative to SRBC. However, see McRee and Hamrick (1977), in which RFR-induced

lower monocyte counts were found for quail from eggs similarly exposed.

REFERENCES: McRee, D. I. and P. E. Hamrick, EXPOSURE OF JAPANESE QUAIL EMBRYOS TO 2.45-GHZ MICROWAVE RADIATION DURING DEVELOPMENT, Radiat. Res., Vol. 71, No. 2, pp. 355-366 (1977).

Huang, A. T. and N. G. Mold
IMMUNOLOGIC AND HEMATOPOIETIC ALTERATIONS BY 2,450-MHZ ELECTROMAGNETIC RADIATION
0197-8462/80/0101-0077, Bioelectromagnetics, Vol. 1, No. 1, pp. 77-87 (1980)

Study type: (8) Immunological, (9) Biochemical/physiological; IN-VIVO; MOUSE

Effect type: Effects of RFR on lymphocyte proliferation, macrophages, hematopoietic cells, and tumor toxicity of killer lymphocytes

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 15 mW/cm.2 and 5 or 30 mW/cm.2

SAR: About 11 mW/g for 15 mW/cm.2

Exposure conditions: Groups of 4 in diamond pattern exposed 30 min/day for 1 to 17 days in far field at constant temperature, humidity, and air-flow rate

AUTHOR ABSTRACT: A biphasic modulation of responsiveness of spleen lymphocytes to mitogens was observed in mice exposed to 2,450-MHz radiation at power densities of 5-15 mW/cm.2 over various periods ranging between one and 17 days. This modulated phenomenon may be explained on the basis of 1) suppression of lymphocyte response by microwave-activated macrophages which persists throughout the entire course of radiation, and 2) concurrent progressive direct stimulation of lymphocytes which culminates around day 9 of exposure. Tumor cytotoxicity of killer lymphocytes from mice exposed to five or nine days of radiation did not appear different from sham controls. The highly proliferative hematopoietic marrow cells were sensitive to microwave radiation. Nine days of exposure to radiation (15 mW/cm.2) reduced the colony-forming units of myeloid and erythroid series by 50%. This observation may offer a new and more sensitive assay for studying biological effects of electromagnetic radiation.

OTHER INFORMATION: Within 60 min after final exposure to 15 mW/cm.2 (30 min/day) or sham exposure, animals were euthanized, spleens were removed, and cells were washed and suspended in culture medium. Phytohemagglutinin (PHA) or Concanavalin A (Con A) (T-cell mitogens) or lipopolysaccharide (LPS) (B-cell mitogen) or no mitogen was added, the cultures were incubated

for 72 hrs, and tritiated thymidine was added 4 hrs before the end of incubation. The cells were then harvested and assessed by labeled thymidine uptake for proliferation. The authors' Table 1 shows the mean uptake of tritiated thymidine for the cultures with PHA, Con A, LPS, and no mitogen, taken from sham and exposed animals after days 2, 4, 5, 7, and 9 of exposure. Also given are the mean uptake ratios of exposed to sham data from concurrently used animals for the same exposure periods. These ratios are plotted against exposure duration for the three mitogens (but not for the no-mitogen data), and show cyclical variations having relative maxima after days 4 and 9. The authors suggest that such cyclical fluctuations could account for the differences in results from various laboratories. Endogenous macrophages were removed from some cultures, yielding improved lymphocyte response to LPS for sham and RFR exposure, with higher response for RFR. Exogenous macrophages from exposed animals were found to diminish response to LPS in unexposed animals. Appropriate antisera were used to characterize spleen subpopulations. After day 9 of RFR exposure to 15 mW/m.2, the B-cell subpopulation was unaltered, but a relative reduction of T-cells was found, accompanied by an increase in the null subpopulation, possibly precursor cells that migrated from the bone marrow. RFR exposure at 15 or 30 mW/cm.2 for 5 days (30 min/day) did not alter the cytotoxic activity of lymphocytes on leukemic cells injected after the last exposure or injected concurrently with exposure at 15 mW/cm.2. In-vitro growth of erythroid and myeloid colonies from animals exposed for 9 days was diminished relative to controls.

FINAL CRITIQUE: The data on mean lymphocyte proliferation versus exposure day for the mitogen-stimulated and no-mitogen cultures from sham-exposed animals (which are presented by the authors in Table 1 but not discussed by them explicitly) show cyclical variations that are evidently due to factors other than RFR, such as circadian rhythms, cyclic changes in female mice, and other uncontrolled factors. These factors undoubtedly contributed, to an undetermined extent, to the numerical results for RFR exposure, rendering it impossible to ascertain the proliferative effects of RFR per se. If, for example, the diurnal response curve differs considerably from mouse to mouse, then taking ratios of mean responses from concurrently used exposed and sham-exposed mice does not remove the non-RFR variations implicit in both sets of data. (In this context, the authors' discussion of the statistical treatment of their data is incomplete and obscure.) For these reasons, all their findings on RFR-induced lymphocyte proliferation, and perhaps their other results as well, are questionable. Cyclical variations may indeed account for differences in

results among various investigators as the authors state, but may be due to uncontrolled non-RFR factors to an unknown extent in the work of these other investigators as well as their own.

Huang, A. T., M. E. Engle, J. A. Elder, J. B. Kinn and T. R. Ward
THE EFFECT OF MICROWAVE RADIATION (2450 MHZ) ON THE MORPHOLOGY AND CHROMOSOMES OF LYMPHOCYTES
Radio Sci., Vol. 12, No. 6S, pp. 173-177 (1977)

Study type: (8) Immunological, (2) Mutagenic and cytogenetic; IN-VIVO; CHINESE HAMSTER

Effect type: RFR-induced blastic transformation, DNA damage, and chromosomal aberrations in blood lymphocytes and bone-marrow cells

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: 0 to 45 mW/cm.2

SAR: 0 to 20.7 W/kg

Exposure conditions: Groups of 5 in far field of source: 1 on axis and the other 4 at corners 30 cm from axis. Exposures were for 15 min/day for 5 consecutive days at each power density in chamber maintained at 22 deg C, 50% humidity

AUTHOR ABSTRACT: Effects of microwave radiation have been examined on blood lymphocytes from Chinese hamsters that were irradiated 15 min each day during five consecutive days by 2450-MHz (CW) energy at power densities from zero to 45 mW/cm.2. Measurements of absorbed energy were accomplished by twin-well calorimetry. One hour after sham or microwave radiation, blood was obtained by orbital hemorrhage and was cultured for one day if unstimulated or for three days if stimulated with phytohemagglutinin (PHA) to induce mitosis. Cultures were terminated with a brief colchicine treatment to arrest cells in metaphase; lymphocytes were then processed for morphological and cytogenetic analysis. Microwave radiation caused a transient, reversible and dose-dependent (albeit curvilinearly related) change in rate of blastic transformation of unstimulated lymphocytes. Rate of transformation was maximum at a power density of 30 mW/cm.2, which is associated with a post-radiation body temperature of 39.3 deg C. However, frequency of mitoses from PHA stimulation was reduced in irradiated samples. Both the enhancement of transformation and the inhibition of mitosis were evident at 5 mW/cm.2. Autoradiography of cells labelled with tritiated-thymidine showed no evidence of radiation-related DNA repair. Cytogenetic analysis demonstrated no difference in chromosomal

aberrations in irradiated and control samples.

OTHER INFORMATION: The power densities used were 0 (sham), 5, 15, 30, or 45 mW/cm.2. Cultures not stimulated with the mitogen PHA exhibited a Transformation Index (percentage of transformed cells relative to the total number) variation with power density in the shape of an inverted U, with maximum value at 30 mW/cm.2, after 24 hr incubation. Subsequent observations over a 5- to 10-day period indicated a gradual return to control values. Cell counts done at the time of blood collection showed no net gain of lymphocytes from other sources such as lymph nodes or the spleen, and no significant changes in leukocyte differential counts, thus supporting the contention that RFR does not cause peripheral lymphocytosis. For cultures stimulated with PHA, the mean value of Mitotic Index (percentage of cells in mitosis relative to the total number of lymphocytes) diminished from 3% for controls to about 0.04% and 0.05% for 30 and 45 mW/cm.2, respectively. However, blastic transformations increased from 8% for controls to 15% for 15 mW/cm.2 (no other data provided). Cultures of hamster bone-marrow cells aspirated from the tibia after exposure were also prepared and analyzed, and chromosomal scoring of these cultures as well as of PHA-stimulated blood lymphocytes showed no significant differences between controls and those from animals exposed at any of the power densities used. In addition, there was no evidence for DNA repair synthesis. Changes of mean rectal temperature after 15-min exposure were -0.2 deg C for 0 mW/cm.2 (controls), +0.2, +0.1, +0.9, and +1.6 deg C for 5, 15, 30, and 45 mW/cm.2, respectively. The Specific Absorption Rate (SAR), measured calorimetrically in dead animals, was 2.3, 6.9, 13.8, and 20.7 W/kg, respectively, at those power densities. Based on a basal metabolic rate of 7.5 W/kg for a 40-g hamster, the authors state that it was possible that the hamsters were heat stressed at the two highest power densities, and that heating from 5 and 15 mW/cm.2 under their experimental conditions can normally be managed and dissipated by a hamster.

FINAL CRITIQUE: The authors do not discuss the rationale for exposing each animal on 5 consecutive days (15 min each day) and for analyzing specimens obtained only after completion of the series, or the possible influence of such an exposure schedule on the findings. They also do not discuss the power density variations among each group of 5 animals exposed concurrently. (Elder and Ali, 1975, cited by the authors, do not do so either.) Their most interesting finding is the inverted-U relationship between the Transformation Index (TI) and the power density for cultures not stimulated with PHA. They suggested that two unspecified controlling mechanisms may

be involved, one operating at the lower power densities and the other that quenches the first at the higher power densities. They state that the quenching mechanism is probably related to the temperature of the animal, but indicate that it was not possible to discern whether the TI increases at 5 and 15 mW/cm.2 were due to heating or some athermal RFR interaction. Because SAR distributions in animals at 2.45 GHz are far from uniform, power densities as low as 5 mW/cm.2 could have produced significant local internal heating. Other unknown factors may have contributed to the results, e.g., the 0.2 deg C decrease in mean rectal temperature after sham exposure is indicative of some non-RFR-induced physiological change. The decrease of the mean Mitotic Index with power density for PHA-stimulated cultures is significant, but equally interesting is that the large scatter of values for the controls (0 to 9%) also diminished rapidly with power density, tending to further confirm that RFR does inhibit mitogen-stimulated mitosis. However, the scatter is still sizable for 5 mW/cm.2, which can be taken as evidence that the effect is of thermal origin.

REFERENCES: J. A. Elder and J. S. Ali, THE EFFECT OF MICROWAVES (2450 MHZ) ON ISOLATED RAT LIVER MITOCHONDRIA, Ann. N.Y. Acad. Sci., Vol. 247, pp. 251-262 (1975)

Smialowicz, R. J., J. B. Kinn, and J. A. Elder
PERINATAL EXPOSURE OF RATS TO 2450-MHZ CW MICROWAVE RADIATION: EFFECTS ON LYMPHOCYTES
0048-6604/79/1112-S022, Radio Sci., Vol. 14, No. 6S, pp. 147-153 (1979)

Study type: (8) Immunological, (3) Teratogenic and developmental abnormalities, (9) Biochemical/physiological; IN-VIVO; RAT

Effect type: RFR-induced weight differences, mitogen-stimulated blast transformations, and blood chemistry changes

Frequency/wavelength: 2.45 GHz

Modulation: CW (11% rms ripple)

Power Densities: 5 mW/cm.2

SAR: 4.7 to 0.7 mW/g

Exposure conditions: Pregnant rats were exposed in arrays of 12 or 8 for 4 hrs/day, 7 days/week. Pups were similarly exposed. Exposures were far-field in a temperature and humidity controlled chamber

AUTHOR ABSTRACT: Groups of rats were exposed IN UTERO from day six of gestation through 40 to 41 days of age for four hours a day in a temperature- and humidity-controlled environment under far-field conditions to 2450-MHz (CW) in an electrically anechoic chamber. An equal number of sham-exposed animals, maintained under the same environmental conditions, served as controls. Two experiments have been performed at this frequency at a power density of 5 mW/cm.2. Specific absorption rates (SARs) for rats of different ages were determined by twin-well calorimetry and were found to range from 0.7 to 4.7 mW/g. Rats were weighed at selected intervals to determine if microwaves affect growth. At 20 to 21 and 40 to 41 days of age, rats were bled and complete blood-cell counts were done. In addition, the IN VITRO blastogenic response of blood and lymph-node lymphocytes were measured by tritiated-thymidine incorporation into DNA following stimulation of cells with T- and B-lymphocyte mitogens. There were significant increases in the mitogen-stimulated response of both T- and B-lymphocytes from irradiated rats. There were no consistent changes, however, in peripheral blood-cell counts after exposure at this frequency. These results indicate that long-term exposure of rats IN UTERO and through early life may result in increased

response of lymphocytes to stimulation with mitogens IN VITRO.

OTHER INFORMATION: On day 6 of pregnancy, rats were concurrently irradiated in an array of 12 in the first experiment and in an array of 8 in the second. Following birth, 4 and 8 male pups of each dam were concurrently irradiated until age 20 days, at which time half were euthanized and the other half were irradiated until age 40 days and then euthanized. Equal numbers of pregnant rats and pups were sham-exposed for controls. Exposures were for 4 hrs/day, 7 days/week. The power density measured at the center of the array was 5 mW/cm.2. The animals were weighed periodically. The 4.7 to 0.7 mW/g SAR range represents the decrease of mean SAR with increase in mean weight (with age) rather than variations among animals at any time. There were no significant differences in mean weight between exposed and control animals at any time. Mean leukocyte counts at age 20 days were significantly lower (p less than 5%) for the exposed group than for controls in the first experiment and were not statistically different in the second. There were no significant differences in leukocyte counts between exposed and control 40-day rats. No changes in erythrocyte counts, hematocrit, or hemoglobin were found in either experiment. Although the authors state that no changes in peripheral lymphocytes or neutrophils were observed, their Table 3 shows a significantly lower value of lymphocytes/mm.3 for the group that exhibited lower leukocyte count. Lymphocytes from blood and cervical lymph nodes were stimulated with the T-cell mitogens phytohemagglutinin (PHA) and concanavalin A (Con A) and with the B-cell mitogens lipopolysaccharide (LPS) and purified protein derivative (PPD). Because of the large variabilities in the results, 14 outlying data points were excluded from the analysis by the authors, based on the use of two statistical tests (Bliss et al., 1956 and Dixon, 1953). The remaining results showed no significant differences in tritiated-thymidine uptake between exposed and control cultures from 20-day rats. However, for the 40-day rats, thymidine uptake after stimulation of nodal lymphocytes with 0.25 microgram/culture of PHA was significantly lower for the exposed group than for the controls in the first experiment and non-significantly higher in the second experiment. Stimulation of blood lymphocytes with 0.5 microgram/culture of PHA yielded higher thymidine uptake for the exposed group in both experiments, but the difference was statistically significant only in the second experiment. Significantly higher thymidine uptakes for the exposed group in the second experiment were also reported for stimulation of nodal lymphocytes with LPS and PPD, but not with Con A. (Corresponding results with these mitogens for the first experiment were not given.)

FINAL CRITIQUE: These results provide no consistent pattern relating higher uptakes of tritiated thymidine in mitogen-stimulated cultures from RFR-exposed rats, perhaps because of the large scattering of the data. The validity of discarding extreme values a posteriori rather than because of foreknowledge that an experimental error had occurred in any specific sample is questionable, and weakens the credibility of the results (whether positive or negative). Perhaps most difficult to understand is that most of the positive results were obtained for the cultures from the 40-day rats, for which the mean SAR had diminished by more than a factor of 2 since age 20 days and by a factor of about 5 since the first few days of exposure. Because of these uncertainties, the results comprise rather weak evidence that perinatal RFR exposure of rats (at this power density and frequency) alters mitogen-stimulated lymphocyte cultures from such rats. Also, if the positive results were to be accepted at face value, it would not be possible to ascertain whether the effects were due to in-utero or postnatal exposure or both, because a group of pups from irradiated dams that was not postnatally exposed had not been included in the study design.

REFERENCES: Bliss, C. I., W. G. Cochran, and J. W. Tukey, A REJECTION CRITERION BASED UPON THE RANGE, Biometrika, Vol. 43, p. 418(1956)

Dixon, W. J., PROCESSING DATA FOR OUTLIERS, Biometrics, Vol. 9, p. 74 (1953)

Stodolnik-Baranska, W.
THE EFFECTS OF MICROWAVES ON HUMAN LYMPHOCYTE CULTURES, In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 189-195 (1974)

Study type: (8) Immunological, (2) Mutagenic and cytogenetic; IN-VITRO; HUMAN LYMPHOCYTES

Effect type: RFR-induced lymphoblastoid transformations and chromosomal abnormalities

Frequency/wavelengths: 2950 MHz

Modulation: 1 microsec pulses, 1200 pps

Power Densities: 7, 20 mW/cm.2 Av

SAR: Not measured

Exposure conditions: Purified peripheral blood lymphocyte suspensions were incubated in TC 199 with 2% inactivated calf serum and exposed to 7 to 20 mW/cm.2 for various durations

REVIEWER SUMMARY: Two series of experiments were performed. In the first, purified peripheral blood lymphocyte suspensions were incubated in TC 199 with 2% inactivated calf serum and exposed to 2950 MHz RFR (1 microsec pulses, 1200 pps) either 4 hrs/day for 3 or 5 days at 7 mW/cm.2 (average power density) or 15 min/day for 3 or 5 days at 20 mW/cm.2, both groups without the mitogen phytohemagglutinin (PHA). In the second series, PHA was added and the following exposures were done with 4 groups: Specimens of Group 1 were incubated for 66 hrs and exposed at 20 mW/cm.2 for 0, 5, 10, 15, 20, or 40 min. Those of Group 2 were incubated for 64 hrs and exposed at 7 mW/cm.2 for either 3 or 4 hrs. Group 3 was exposed 4 hrs/day for 3 days at 7 mW/cm.2. Those of Group 4 were exposed for 10 min at 20 mW/cm.2 following 0, 59, 64 (?), 70, or 71.5 hrs of incubation. The author tabulated only the results for 20 mW/cm.2 with PHA (Groups 1 and 4). Those for Group 1 showed no significant changes in percentage of blastoid forms, but a decrease of the percentage of lymphocytes with exposure duration. The mitotic index (MI) for this group changed from 12.0% for no RFR exposure to: 13.7 for 5 min of exposure; 17.2 and 17.9 for 10 and 15 min, respectively; and 23.5 and 25.0 for 20 and 40 min, respectively. The author stated that similar results were obtained for Group 2 (at 7 mW/cm.2). The results for Group 4 indicated increases in MI from 12.0 for 0 hrs of incubation to 15.6 for 59 hrs and to 17.0 for 64 hrs of

incubation. Decreases to 10.2 and 11.0 were obtained for 70 and 71.5 hrs of incubation, respectively. The author also reported increases in chromosomal aberrations with exposure duration for Group 1, including stickiness of chromosomal arms, dicentrics, hypoploidy, hyperploidy, and chromatid breaks, illustrating each type with a micrograph. She stated that alterations in chromosomal morphology suggesting changes in spiralization were the most unusual finding. Regarding exposures of specimens without PHA, she stated: "It should be stressed that irradiation of lymphocytes without PHA addition induces the appearance of blastoid forms and macrophage-like cells," illustrated by one micrograph, but gave no data.

OTHER INFORMATION: The author stated that the temperature of the medium remained constant (at 37 deg C) during 4 hrs of exposure at 7 mW/cm.2, and that at 20 mW/cm.2, the temperature increased by 0.5 deg C after 15 min and by 1 deg C after 20 min. The author first reported such work in less detail in Stodolnik-Baranska, 1967.

FINAL CRITIQUE: It is difficult to evaluate this paper because the results of some of the experiments mentioned are not presented, e.g., the numerical values obtained at 7 mW/cm.2 and those for the cultures without PHA. Also, the purpose of performing exposures on the latter cultures is not clear. From the results at 20 mW/cm.2 with PHA-containing cultures, there was a trend toward increase of mitotic index with exposure duration, decrease of the percentage of lymphocytes with duration, but no significant changes in the percentage of blastoid forms. Thus, it is difficult to understand how the results suggest that microwaves may have mutagenic effects, as stated by the author. The changes found may have been of thermal origin rather due to any intrinsic effect of RFR, because the author indicated that exposure of cultures at 20 mW/cm.2 for 20 minutes resulted in a 1 deg C rise in the cultures, i.e., the temperature of the cultures was not effectively controlled.

REFERENCES: Stodolnik-Baranska, W., LYMPHOBLASTOID TRANSFORMATION OF LYMPHOCYTES IN VITRO AFTER MICROWAVE IRRADIATION, Nature, Vol. 214, pp. 102-103 (1 April 1967).

Wiktor-Jedrzejczak, W., A. Ahmed, P. Czerski, W. M. Leach, and K. W. Sell
EFFECT OF MICROWAVES (2450-MHZ) ON THE IMMUNE SYSTEM IN MICE: STUDIES OF NUCLEIC ACID AND PROTEIN SYNTHESIS,
0197-8462/80/0102-0161, Bioelectromagnetics, Vol. 1, No. 2, pp. 161-170 (1980)

Study type: (8) Immunological; IN-VIVO; MOUSE

Effect type: RFR-induced changes in DNA, RNA, and protein synthesis

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: Not given; 0.6 W forward power in waveguide

SAR: 12-15 mW/g

Exposure conditions: Single or 3 exposures each of 30 min duration in a waveguide under constant temperature, humidity and airflow rate through the waveguide

AUTHOR ABSTRACT: CBA/J adult male mice were given single or triple exposures to 2450-MHz microwaves in an environmentally controlled waveguide facility. The average absorbed dose rate for a single exposure varied from 12 to 15 mW/g. Sham-exposed mice served as controls. Lymphoid cells were collected and tested for metabolic activity on days 3, 6, and 9 following a single exposure, and on days 9, 12, and 16 following triple exposures on days 0, 3 and 6. Cells were cultured in vitro for four hours to seven days before their metabolic rates were assayed. Under these conditions, microwaves failed to produce any detectable change in deoxyribonucleic acid (DNA), ribonucleic acid (RNA), and protein synthesis, as measured by the incorporation of methyl(tritium)-thymidine (DNA substrate), tritiated uridine (RNA substrate), and tritiated leucine(protein substrate) by spleen, bone marrow, and peripheral blood lymphocytes (PBL) in vitro. These data suggest that microwave-induced increases in the frequency of complement-receptor (CR)- or surface-immunoglobulin (sIg)-bearing cells were not associated with a concomitant increase in cell proliferation and/or protein synthesis, and favor the concept that microwaves under these conditions stimulate already existing B-cell precursors for maturation.

OTHER INFORMATION: The objective of this study was to determine whether the increased frequency of

complement-receptor positive (CR+) B-lymphocytes from IN-VIVO exposure of mice to 2.45 GHz at whole-body SARs of about 14 mW/g, found in their previous study (Wiktor-Jedrzejczak et al., 1977), is associated with elevated cell proliferation in the spleen or bone marrow. Mice were exposed individually to RFR in groups of 6 per experiment, with 6 sham-exposed mice serving as controls. Exposures were for 30 min/session, either for a single session or 3 sessions, 1 per day on days 0, 3, and 6. For the mice given single sessions, peripheral blood and spleen and bone-marrow cells were obtained on days 3, 6, or 9 after exposure, and were assayed for incorporation of tritiated thymidine, uridine, and leucine (without mitogen stimulation) in cultures incubated for 4 hrs, 3 days, and 5 days. For mice exposed on each of days 0, 3, and 6, the specimens were obtained on days 9, 12, and 16 (i.e., on days 3, 6, and 10 after the third exposure), and were similarly assayed in cultures incubated for 4 hrs and 3, 5, and 7 days. No significant differences, between exposed and control groups, in the incorporation of thymidine, uridine, or leucine were found in any of the cultures studied. The frequency of CR+ splenocytes was also determined for each group of mice, and the authors stated that significant increases were found in all cultures from the groups given a single RFR session except in the cultures taken on day 3 after exposure, but they presented no data. They concluded: "The present study, along with previous studies, show that the increased number of CR+ splenocytes observed following exposure of mice to 2450-MHz CW microwaves is not associated with significant changes in the rate of DNA, RNA, or protein synthesis by spleen, bone marrow, or peripheral blood lymphocytes."

FINAL CRITIQUE: The results of this study negate the hypothesis that increases in CR+ due to IN-VIVO exposures of mice to RFR are associated with elevated cell proliferation. This finding appears to be at variance with the results of Stodolnik-Baranska (1974, 1967) and Czerski (1975). Stodolnik-Baranska had exposed mitogen (PHA)-stimulated human-lymphocyte cultures to RFR (IN VITRO), and found no significant changes in percentages of blastoid forms, but observed significant decreases in percentages of lymphocytes and increases in mitotic index with exposure duration. Czerski's results with IN-VITRO RFR exposure of PHA-stimulated human lymphocyte cultures, though poorly reproducible, indicated the occurrence of blastic transformation. In addition, he reported increases in percentages of blast cells for mice exposed (IN VIVO) to RFR followed by immunization with sheep red blood cells as compared with mice that were not exposed but were immunized. The authors of the present paper indicated that because of differences in exposure conditions,

species, and evaluation techniques, their findings may not necessarily contradict those of Stodolnik-Baranska (1974, 1967) and Czerski (1975). This statement is probably true for these IN-VITRO studies but perhaps not for the IN-VIVO mice studies of Czerski, thereby leaving the question of RFR-induced cell proliferation open. The alternative hypothesis suggested by the authors is that the observed increases in CR+ cells indicate that RFR stimulates B-cell precursors to express CR earlier than normally occurs without RFR, i.e., that RFR induces earlier maturation of B cells rather that increases their total numbers. Further work appears to be necessary to test this new hypothesis.

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Wiktor-Jedrzejczak, W., A. Ahmed, P. Czerski, W. M. Leach, and K. W. Sell, IMMUNE RESPONSE OF MICE TO 2450-MHZ MICROWAVE RADIATION: OVERVIEW OF IMMUNOLOGY AND EMPIRICAL STUDIES OF LYMPHOID SPLENIC CELLS, Radio Sci., Vol. 12, No. 6S, pp. 209-219 (1977)

Wiktor-Jedrzejczak, W., A. Ahmed, P. Czerski, W. M. Leach, and K. W. Sell
IMMUNE RESPONSE OF MICE TO 2450-MHZ MICROWAVE RADIATION: OVERVIEW OF IMMUNOLOGY AND EMPIRICAL STUDIES OF LYMPHOID SPLENIC CELLS
Radio Sci., Vol. 12, No. 6S, pp. 209-219 (1977)

Study type: (8) Immunological; IN-VIVO; MOUSE

Effect type: Effects of RFR on splenic T and B lymphocytes

Frequency/wavelength: 2.45 GHz

Modulation: CW

Power Densities: Not given; 0.6 W forward power in waveguide

SAR: About 14 mW/g

Exposure conditions: Single or 3 exposures each of 30 min duration in a waveguide under constant temperature, humidity, and airflow rate through the waveguide

AUTHOR ABSTRACT: Male CBA/J mice were exposed to 2450-MHz microwave radiation (each exposure: 30 minutes at an averaged dose rate near 14 mW/g). The mice were tested later to determine effects of the radiation on (1) the relative frequency of T and B cells; (2) the functional capacity of spleen cells from irradiated mice to respond to T- and B-cell-specific membrane stimuli; and (3) the ability to respond to sheep red blood cells and dinitrophenyl-lyslyl-Ficoll. Results demonstrated that microwave radiation has weak stimulatory effects on B- but not T-lymphoid cells in the spleen. Single exposures to radiation produced an increase in the incidence of cells with complement receptor on the cell's surface. Three exposures to radiation induced increases in the total number of splenic cells, in the incidence of immunoglobin-positive cells, and in the incidence of complement receptor-positive cells. The total number of T cells was unaffected by single to triple exposures of mice to the radiation.

OTHER INFORMATION: Mice were exposed individually to RFR in groups of 6 per experiment, with 6 sham-exposed mice serving as controls. Exposures were for 30 min/session, either 1 session or 3 sessions, 1 per day on days 0, 3, and 6. For groups not studied with mitogens, the spleens were removed 6 days after the last exposure session. The results for single exposures of these groups showed no significant differences in

subpopulations of theta-positive T-cells or Ig-positive B-cells between exposed and control groups, but the exposed groups yielded significantly larger subpopulations of complement-receptor-positive (CR+) B-cells. For those given 3 exposures, significantly larger subpopulations of Ig-positive as well as CR+ B-cells were found. For assays of mitogen-stimulated splenic cells, the spleens were removed 3, 6, or 9 days after the last exposure. The T-cell mitogens used were phytohemagglutinin-P (PHA-P) and concanavalin A (con A); the B-cell mitogens were dextran sulfate (DxS), lipopolysaccharide (LPS), polyinosinic polycytidylic acid (Poly.I.C), and purified protein derivative (PPD); pokeweed mitogen (PWM), which induces proliferation of both T- and B-cells, was used as a nonspecific mitogen. Cultures were incubated at 37 deg C for 72 hrs, and 18 hrs prior to harvest, each culture was given 1 microCurie of tritiated thymidine, the uptake of which is a measure of DNA synthesis. The results for the T-cell mitogens with single and three-session exposures showed no significant differences (at less than the 5% level) in thymidine uptake between exposed and control groups. However, for the animals given a single exposure, significant increases of thymidine uptake were reported: for the mitogen PPD in cultures harvested 3, 6, and 9 days after the exposure; Poly.I.C in cultures harvested 3 and 6 days after the exposure, but not in those harvested 9 days postexposure; and for LPS only in cultures harvested 9 days after exposure. For the animals given 3 exposures, significantly higher thymidine uptake was found only for LPS, in cultures harvested 12 days after the first exposure (i.e., 6 days after the last exposure) but not in those taken 9 days after the first exposure (3 days after the last exposure). In another set of experiments, groups of 4 mice were immunized with the T-dependent antigen sheep red blood cells (SRBC) or the T-independent antigen dinitrophenyl-lyslyl-Ficoll (DNP-lys-Ficoll), or with saline for control. Groups were then given 30-min exposures or sham-exposures on days 1, 2, and 3. Their spleens were removed on day 4 and assayed for antibody formation by counting plaque-forming cells (PFC). There were no significant differences in PFC between RFR- and sham-exposed groups pretreated with DNP-lys-Ficoll or saline, but significantly lower values were obtained for the RFR-exposed group with SRBC.

FINAL CRITIQUE: The statistical treatment of the data presented is not clear in that the authors did not state whether they used the 1-tailed or 2-tailed t-test. Although many of the t values would be significant at less than the 5% level under either test, some differences labeled by them as significant may not be or conversely. Specifically, reviewer calculations of t indicate that the investigators apparently

used the 1-tailed test for all data except for those pertaining to subpopulations of T- and B-cells without mitogen stimulation, for which they used the 2-tailed test (without indicating the justification for either usage). Had they used the 1-tailed test for the latter, then the mean of the theta-positive T-cells for the 3-exposure experiment would be significantly lower for the exposed group than the control group. Conversely, if they had used the 2-tailed test on all the other data presented, then the single-exposure differences for Poly.I.C after 3 days and those for PPD after 3 and 9 days would not be significant. Apart from such statistical considerations, the conclusion that such RFR exposures of mice have weak stimulatory effects on B-cells but not on T-cells of the spleen appears to be qualitatively valid. However, clear patterns in the data are difficult to discern and apparently contradictory results were obtained in a few cases, e.g., in the LPS work. At the whole-body SARs used, any effects found could be characterized as thermal in origin. (The small changes in rectal temperature are not suitable indices of local internal temperatures.)

(9) BIOCHEMICAL/PHYSIOLOGICAL

List of Analyses

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In ABSTRACTS OF OPEN SYMPOSIUM ON THE BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, Helsinki, Finland (1978) (See "Nervous System (EEG and EP)" for analysis.)

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Health Phys., Vol. 33, pp. 23-33 (1977) (See "Immunological" for analysis.)

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(9) BIOCHEMICAL/PHYSIOLOGICAL

List of Analyses (continued)

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EFFECTS OF REPEATED EXPOSURE TO 148-MHZ RADIO WAVES ON GROWTH AND HEMATOLOGY OF MICE
0048-6604/79/1112-S026, Radio Sci., Vol. 14, No. 6S, pp. 173-179 (1979)

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THERMAL AND ENDOCRINOLOGICAL EFFECTS OF PROTRACTED IRRADIATION OF RATS BY 2450-MHZ MICROWAVES
Radio Sci., Vol. 12, No. 6S, pp. 147-156 (1977) (See "Endocrinological" for analysis.)

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(9) BIOCHEMICAL/PHYSIOLOGICAL

List of Analyses (continued)

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RESPONSES OF THE MOUSE TO MICROWAVE RADIATION DURING ESTROUS CYCLE AND PREGNANCY
Radiat. Res., Vol. 62, pp. 225-241 (1975)
(See "Teratogenic and Developmental Abnormalities" for analysis.)

[1]Sadchikova, M. N.
CLINICAL MANIFESTATIONS OF REACTIONS TO MICROWAVE IRRADIATION IN VARIOUS OCCUPATIONAL GROUPS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 261-267 (1974) (See "Epidemiologic" for analysis.)

[1]Siekierzynski, M.
A STUDY OF THE HEALTH STATUS OF MICROWAVE WORKERS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 273-280 (1974) (See "Epidemiologic" for analysis.)

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PERINATAL EXPOSURE OF RATS TO 2450-MHZ CW MICROWAVE RADIATION: EFFECTS ON LYMPHOCYTES
0048-6604/79/1112-S022, Radio Sci., Vol. 14, No. 6S, pp. 147-153 (1979) (See "Immunological" for analysis.)

Analyses under

(9) BIOCHEMICAL/PHYSIOLOGICAL

Lin, J. C., J. C. Nelson, and M. E. Ekstrom
EFFECTS OF REPEATED EXPOSURE TO 148-MHZ RADIO WAVES ON GROWTH AND HEMATOLOGY OF MICE
0048-6604/79/1112-S026, Radio Sci., Vol. 14, No. 6S, pp. 173-179 (1979)

Study type: (9) Biochemical/physiological, (8) Immunological; IN VIVO; MOUSE

Effect type: RFR-induced changes in animal weight, hematocrit, hemoglobin, leukocyte and erythrocyte counts, and differential lymphocyte and segmented neutrophil counts

Frequency/wavelength: 148 MHz

Modulation: CW

Power Densities: 0.5 mW/cm.2 Av (63.25 V/m Pk)

SAR: 0.013 W/kg

Exposure conditions: 1 hr/day, 5 days/week for 10 weeks in a TEM exposure chamber (Crawford cell) beginning on the 4th to 7th day post partum. Controls were sham-exposed. Ambient temperature and humidity were between 25 and 35 deg C and 35 to 65%, respectively.

AUTHOR ABSTRACT: The effects of repeated exposure of mice to RF energy (148 MHz)were investigated. Mice were exposed at 0.5 mW/cm.2 (63.25 V/m) in a TEM exposure chamber. They were irradiated for one hour a day, five days a week, beginning on the 4th to the 7th day postpartum, for ten weeks. The mice were divided into two equal groups to serve as sham-irradiated controls and irradiated subjects. The animals were weighed daily from the beginning of irradiation treatments for ten weeks, and weekly thereafter. The results indicate that the formed elements in the blood of the mouse are not affected by exposure to low-level VHF fields. The means of body mass of the irradiated and control animals were comparable, which indicates that the animals remained in comparably good health during irradiation.

OTHER INFORMATION: Totals of 87 and 88 mice were used for sham-exposure and RFR-exposure, respectively. The animals were weighed daily from the beginning of exposure through the 10-week exposure period and weekly thereafter up to 600 days of age. Differences in weights were not statistically significant at the 5% level. Blood (less than 45 microliters per sampling) was drawn from tail vessels at 28 and 70 days of age (during

the 10-week exposure period) and at 100, 250, 300, 360, and 600 days of age (post-exposure). No statistically significant differences between RFR-exposed and sham-exposed groups were found for hematocrit, hemoglobin, leukocyte counts or erythrocyte counts. In addition, there were no significant differences in differential blood-cell counts, but only the data on lymphocytes and segmented neutrophils were presented. Thus, all the findings of this investigation were negative.

FINAL CRITIQUE: Based on the low SAR (0.013 mW/g) for a mouse at this frequency and incident power density, RFR-induced thermal effects would not be expected, and the absence of any of the effects sought tends to counter the hypothesis of nonthermal RFR effects.

(10) CELLULAR

List of Analyses

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DETERMINATION OF A THERMAL EQUIVALENT OF MILLIMETER MICROWAVES IN LIVING CELLS
0022-2739/79/0012-0307, J. Microwave Power, Vol. 14, No. 4, pp. 307-312 (1979)

[2] Dietzel, F.
EFFECTS OF ELECTROMAGNETIC RADIATION ON IMPLANTATION AND INTRAUTERINE DEVELOPMENT OF THE RAT
Ann. N.Y. Acad. Sci., Vol. 247, pp. 367-376 (1975)
(See "Teratogenic and Developmental Abnormalities" for analysis.)

Analyses under

(10) CELLULAR

Dardalhon, M., D. Averbeck, and A. J. Berteaud
DETERMINATION OF A THERMAL EQUIVALENT OF MILLIMETER MICROWAVES IN LIVING CELLS
0022-2739/79/0012-0307, J. Microwave Power, Vol. 14, No. 4, pp. 307-312 (1979)

Study type: (10) Cellular, (3) Teratogenic and developmental abnormalities, (16) Physical methods/dosimetry

Effect type: RFR-induced alterations of cellular growth and genetic insult

Frequency/wavelength: 70-75 GHz

Modulation: CW

Power Densities: 6, 15, and 60 mW/cm.2 at horn face

SAR: Not given

Exposure conditions: RFR exposure for 180 min at 20 deg C; conventional heating at 30, 42, 47, or 52 deg C for 330 min

AUTHOR ABSTRACT: Recent microwave experiments have shown frequency dependent influences on the growth rate of bacteria. To determine whether microwaves are able to affect growth (or to induce lesions in cellular DNA of yeast cells), experiments were performed with millimeter microwaves at frequencies between 70 and 75 GHz. Saccharomyces cerevisiae cells were irradiated on millipore filter discs placed on agar plates in open petri dishes. A diploid strain of yeast (D-5, Zimmermann) that is sensitive to genetic insult was used to study the effects of temperature and of microwave irradiation on cell survival, induction to mitotic recombination, and induction of cytoplasmic "petite" mutations. No evidence of altered survival, impared (sic) function, or structural injury was seen at either frequency, even at power densities as high as 60 mW/cm.2. Conventional heating had no deleterious effects until temperatures of specimens exceeded 50 deg C. In addition, two haploid strains of yeast of opposite mating type were compared with respect to temperature and microwave treatment for formation of zygotes. The elevation of temperature due to the microwave treatment at 60 mW/cm.2 and 2 mm distance was estimated to correspond to 3 deg C.

OTHER INFORMATION: The cited power density values were measured or calculated for the face of the horn antenna used for irradiation. The distance from the horn face to the specimen was then specified as either 2 or 10 mm, but the

actual power densities at the specimen were not stated. To estimate temperature increments at 10 mm from the horn face, with 60 mW/cm.2 at the latter, water evaporation from solidified agar was measured over 180 min and compared with evaporation due to temperatures of 20, 30, and 37 deg C produced by conventional heating. The water loss rate from RFR was comparable to that at 20 deg C, and the temperature elevation of specimens under such RFR exposure was estimated to be about 3 deg C. Survival rates of cells exposed at 10 mm to 15 or 60 mW/cm.2 of 70.5 or 73 GHz RFR for 180 min were unaltered. There was also no apparent effect on nuclear DNA. However, with conventional heating to 52 deg C, cell survival decreased strongly and induction of altered colonies and of cytoplasmic "petite" mutations increased. The ratio of zygote formation between two haploid yeast strains of opposite mating types was measured for 6, 15, and 60 mW/cm.2 at 2 and 10 mm from the horn. The ratio increased only slightly with power density at 10 mm but sharply above 6 mW/cm.2 at 2 mm. Conventional heating to 20, 23, 30, and 37 deg C indicated even sharper increases in this ratio above about 23 deg C. The authors conclude that exposure to millimeter waves under their conditions does not induce lesions and genetic effects in cellular DNA, whereas intense conventional heating does.

FINAL CRITIQUE: Power densities at the specimen are not given and cannot be estimated because the dimensions of the horn used for irradiation are not specified. However, the result that irradiation of specimens for 180 min at 10 mm from the horn with a power density of 60 mW/cm.2 produced a temperature rise of about 3 deg C is probably valid. Thus, the findings of no effects for a temperature rise of this magnitude is also valid. It should be noted that depth of penetration of the RFR into the specimens was probably not an important factor because of the presumed thinness of each specimen.

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(11) MECHANISMS OF INTERACTION

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SENSITIVITY OF CALCIUM BINDING IN CEREBRAL TISSUE TO WEAK ENVIRONMENTAL FIELDS OSCILLATING AT LOW FREQUENCY
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(See "Nervous System (Calcium efflux)" for analysis.)

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(13) MEDICAL APPLICATIONS

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Ann. N.Y. Acad. Sci., Vol. 247, pp. 367-376 (1975) (See "Teratogenic and Developmental Abnormalities" for analysis.)

(14) REVIEW

List of Analyses

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CLINICAL MANIFESTATIONS OF REACTIONS TO MICROWAVE IRRADIATION IN VARIOUS OCCUPATIONAL GROUPS
In P. Czerski et al. (eds.), BIOLOGIC EFFECTS AND HEALTH HAZARDS OF MICROWAVE RADIATION, Polish Medical Publishers, Warsaw, pp. 261-267 (1974) (See "Epidemiologic" for analysis.)

(16) PHYSICAL METHODS/DOSIMETRY

List of Analyses

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DETERMINATION OF A THERMAL EQUIVALENT OF MILLIMETER MICROWAVES IN LIVING CELLS
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